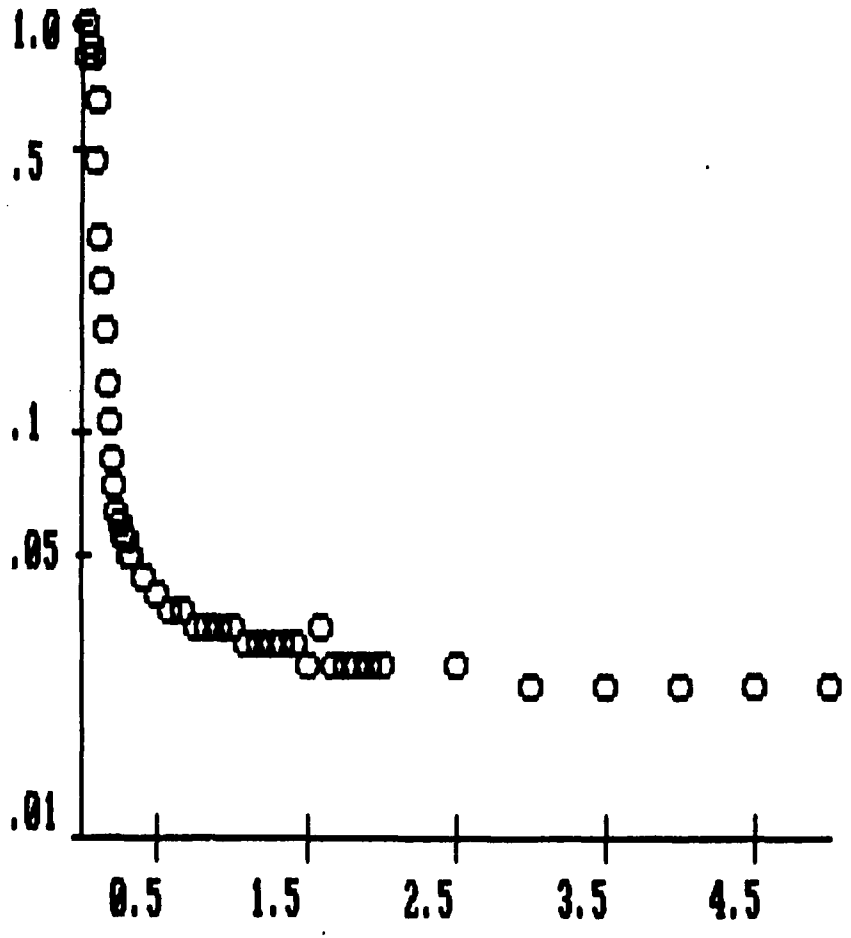
Summit National Site -- MW-11

TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
1.8	3	3.00	1
1.998	2.49	2.49	.8300001
3	2.63	2.63	.8766668
3.996	2.47	2.47	.8233334
4.998	1.39	1.39	.4633334
6	1.97	1.97	.6566667
6.996	.92	0.92	.3066667
7.998	.71	0.71	.2366667
9	.53	0.53	.1766667
9.996	.4	0.40	.1333333
10.998	.32	0.32	.1066667
12	.26	0.26	8.666667E-02
12.996	.22	0.22	7.333334E-02
13.998	.19	0.19	6.333333E-02
15	.18	0.18	6.000001E-02
15.996	.17	0.17	5.666667E-02
16.998	.16	0.16	5.333334E-02
18	.16	0.16	5.333334E-02
18.996	.15	0.15	5.000001E-02
19.998	.15	0.15	5.000001E-02
25.002	.13	0.13	4.333334E-02
30	.12	0.12	.04
34.998	.11	0.11	3.666667E-02
40.002	.11	0.11	3.666667E-02
45	.1	0.10	3.333334E-02

UNCONFINED AQUIFER

K = 0.3E-02 cm/sec
  = 67.9 gpd/ft2
  = 0.1E-03 ft/sec
  = 9.1 ft/day

REGRESSION COEFFICIENT = -.8152988



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

MW-11 (cont.)

960	3.5	3.50	.9615384
1080	3.48	3.48	.9560439
1200	3.46	3.46	.9505494
1320	3.45	3.45	.9478022
1440	3.43	3.43	.9423077
1560	3.41	3.41	.9368132
1680	3.4	3.40	.9340659
1800	3.38	3.38	.9285714
1920	3.35	3.35	.9203296
2040	3.34	3.34	.9175823
2160	3.33	3.33	.9148351
2280	3.31	3.31	.9093406
2400	3.3	3.30	.9065933
2520	3.28	3.28	.9010989
2640	3.26	3.26	.8956044
2760	3.25	3.25	.8928571
2880	3.23	3.23	.8873626
3000	3.22	3.22	.8846154
3120	3.2	3.20	.8791209
3240	3.19	3.19	.8763736
3360	3.17	3.17	.8708791
3480	3.15	3.15	.8653846
3600	3.13	3.13	.8598902
3720	3.12	3.12	.8571428
3840	3.1	3.10	.8516483
3960	3.09	3.09	.8489011
4080	3.07	3.07	.8434066
4200	3.06	3.06	.8406593

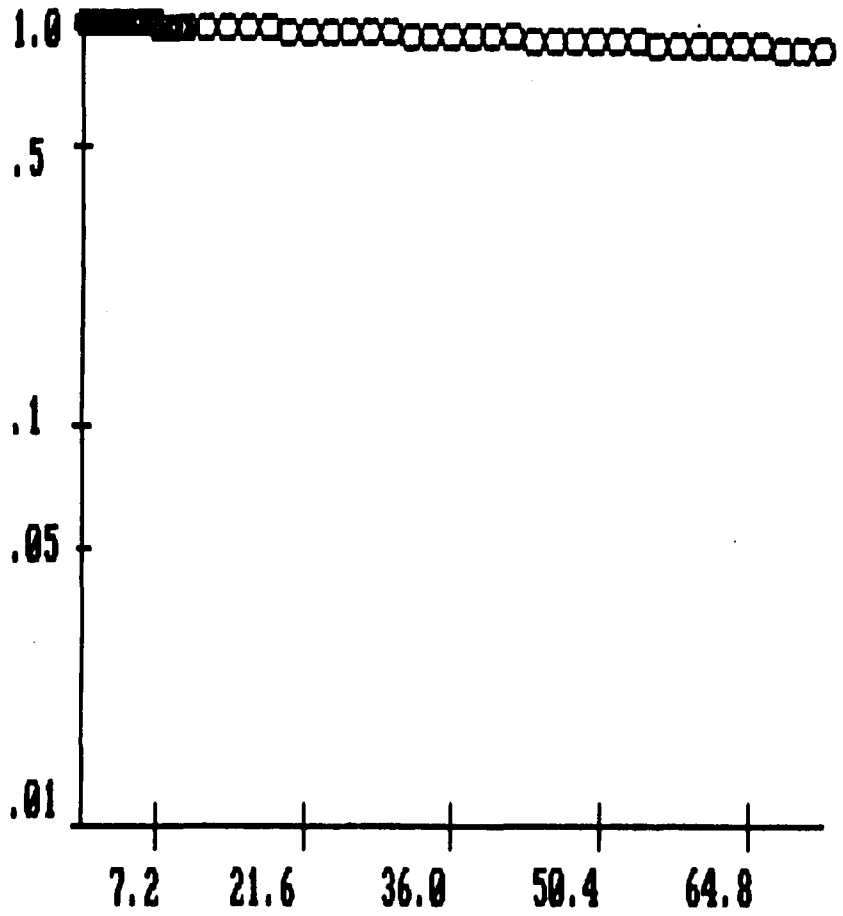
CONFINED AQUIFER, FULLY PENETRATING CONDITION

K = 0.6E-05 cm/sec
= 0.1 gpd/ft2
= 0.2E-06 ft/sec
= 0.0 ft/day

REGRESSION COEFFICIENT = -.9996982

[illegible] National Site -- MW-12

TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
7.998	3.64	3.64	1
9	3.64	3.64	1
9.996	3.64	3.64	1
10.998	3.64	3.64	1
12	3.64	3.64	1
12.996	3.64	3.64	1
13.998	3.64	3.64	1
15	3.64	3.64	1
15.996	3.64	3.64	1
16.998	3.64	3.64	1
18	3.64	3.64	1
18.996	3.64	3.64	1
19.998	3.64	3.64	1
25.002	3.64	3.64	1
30	3.64	3.64	1
34.998	3.64	3.64	1
40.002	3.64	3.64	1
45	3.64	3.64	1
49.998	3.64	3.64	1
55.002	3.64	3.64	1
60	3.64	3.64	1
64.998	3.64	3.64	1
70.002	3.64	3.64	1
75	3.64	3.64	1
79.998	3.64	3.64	1
84.996	3.64	3.64	1
90	3.64	3.64	1
94.998	3.64	3.64	1
100.002	3.64	3.64	1
105	3.64	3.64	1
109.998	3.64	3.64	1
115.002	3.64	3.64	1
120	3.63	3.63	.9972528
150	3.63	3.63	.9972528
180	3.62	3.62	.9945054
210	3.62	3.62	.9945054
240	3.61	3.61	.9917582
270	3.61	3.61	.9917582
300	3.6	3.60	.989011
330	3.6	3.60	.989011
360	3.6	3.60	.989011
390	3.59	3.59	.9862637
420	3.59	3.59	.9862637
450	3.58	3.58	.9835164
480	3.58	3.58	.9835164
510	3.57	3.57	.9807692
540	3.57	3.57	.9807692
570	3.56	3.56	.978022
600	3.56	3.56	.978022
720	3.54	3.54	.9725274
840	3.52	3.52	.9670329



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

MW-12 (cont.)

Summit National Site -- MW-13

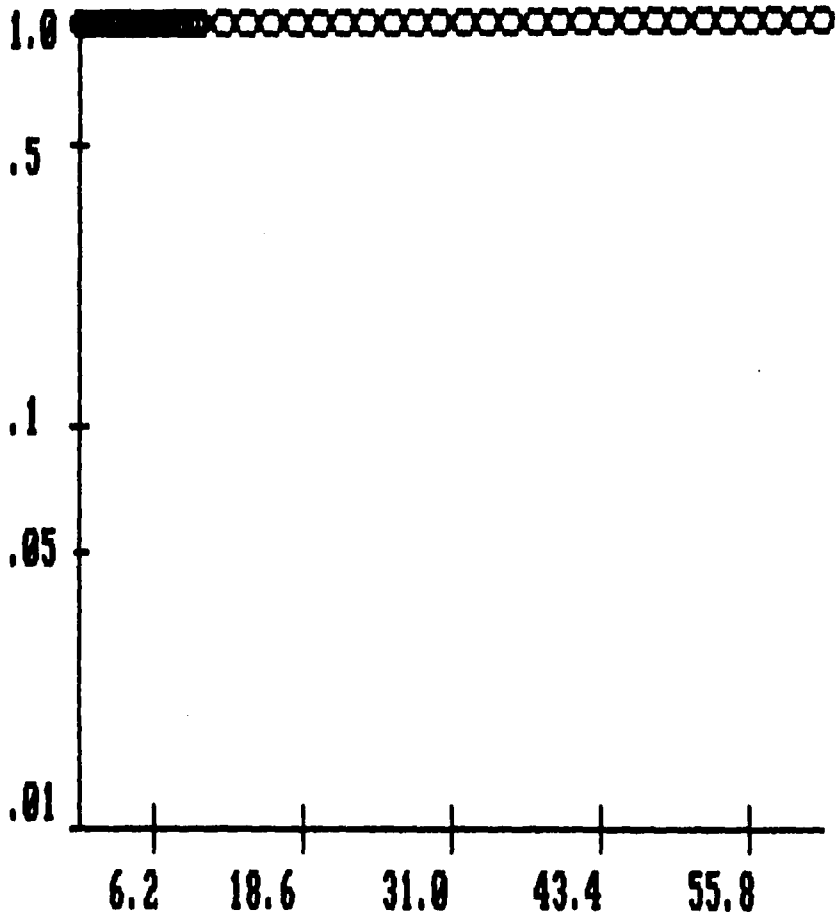
TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
6.996	3.61	3.61	1
7.998	3.6	3.60	.99723
9	3.6	3.60	.99723
9.996	3.61	3.61	1
10.998	3.61	3.61	1
12	3.61	3.61	1
12.996	3.61	3.61	1
13.998	3.61	3.61	1
15	3.61	3.61	1
15.996	3.61	3.61	1
16.998	3.61	3.61	1
18	3.61	3.61	1
18.996	3.61	3.61	1
19.998	3.61	3.61	1
25.002	3.61	3.61	1
30	3.61	3.61	1
34.998	3.61	3.61	1
40.002	3.61	3.61	1
45	3.61	3.61	1
49.998	3.61	3.61	1
55.002	3.61	3.61	1
60	3.61	3.61	1
64.998	3.61	3.61	1
70.002	3.61	3.61	1
75	3.61	3.61	1
79.998	3.61	3.61	1
84.996	3.61	3.61	1
90	3.61	3.61	1
94.998	3.61	3.61	1
100.002	3.61	3.61	1
105	3.61	3.61	1
109.998	3.61	3.61	1
115.002	3.61	3.61	1
120	3.61	3.61	1
150	3.61	3.61	1
180	3.61	3.61	1
210	3.61	3.61	1
240	3.61	3.61	1
270	3.61	3.61	1
300	3.61	3.61	1
330	3.61	3.61	1
360	3.61	3.61	1
390	3.61	3.61	1
420	3.61	3.61	1
450	3.6	3.60	.99723
480	3.6	3.60	.99723
510	3.6	3.60	.99723
540	3.6	3.60	.99723
570	3.6	3.60	.99723
600	3.6	3.60	.99723
720	3.6	3.60	.99723

840	3.6	3.60	.99723
960	3.6	3.60	.99723
1080	3.6	3.60	.99723
1200	3.6	3.60	.99723
1320	3.6	3.60	.99723
1440	3.6	3.60	.99723
1560	3.6	3.60	.99723
1680	3.6	3.60	.99723
1800	3.6	3.60	.99723
1920	3.6	3.60	.99723
2040	3.6	3.60	.99723
2160	3.6	3.60	.99723
2280	3.6	3.60	.99723
2400	3.59	3.59	.9944598
2520	3.59	3.59	.9944598
2640	3.6	3.60	.99723
2760	3.59	3.59	.9944598
2880	3.59	3.59	.9944598
3000	3.59	3.59	.9944598
3120	3.59	3.59	.9944598
3240	3.6	3.60	.99723
3360	3.58	3.58	.9916398
3480	3.57	3.57	.9889197
3600	3.59	3.59	.9944598
3720	3.6	3.60	.99723

CONFINED AQUIFER, FULLY PENETRATING CONDITION

K = 0.2E-06 cm/sec
= 0.0 gpd/ft2
= 0.6E-08 ft/sec
= 0.0 ft/day

REGRESSION COEFFICIENT = -.8481028



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

*M-13 (cont.)

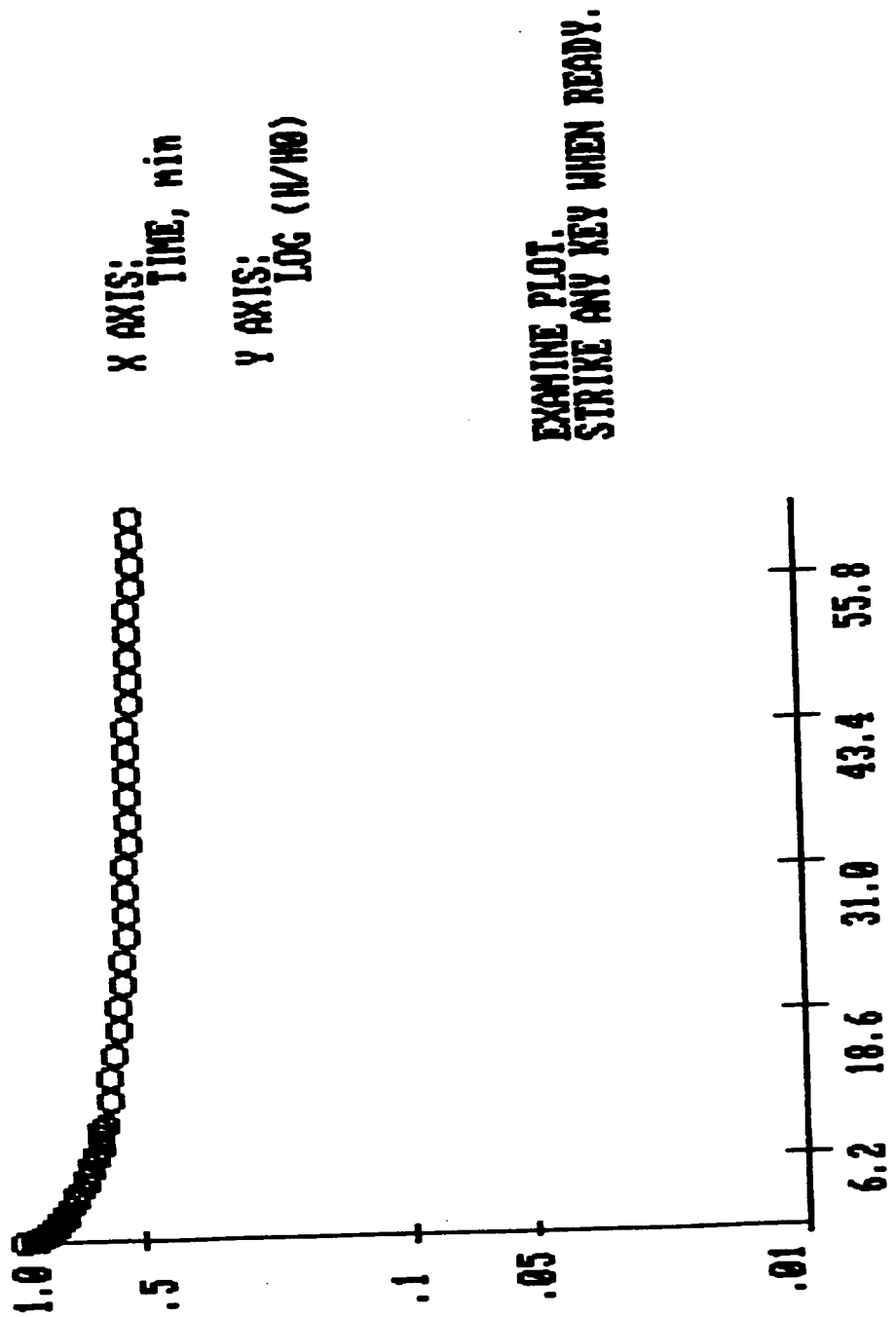
Summit National Site -- MW-14

TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
60	3.21	3.21	.8492064
64.998	3.19	3.19	.9439154
70.002	3.17	3.17	.8386245
75	3.16	3.16	.8359789
79.998	3.13	3.13	.8280425
84.996	3.12	3.12	.8253968
90	3.1	3.10	.8201059
94.998	3.08	3.08	.8148149
100.002	3.06	3.06	.8095238
105	3.05	3.05	.8068783
109.998	3.03	3.03	.8015873
115.002	3.02	3.02	.7989418
120	3	3.00	.7936508
150	2.93	2.93	.7751323
180	2.86	2.86	.7566138
210	2.8	2.80	.7407408
240	2.74	2.74	.7248678
270	2.69	2.69	.7116403
300	2.64	2.64	.6984128
330	2.6	2.60	.6878307
360	2.55	2.55	.6746032
390	2.51	2.51	.6640212
420	2.48	2.48	.6560847
450	2.45	2.45	.6481482
480	2.42	2.42	.6402117
510	2.4	2.40	.6349207
540	2.38	2.38	.6296297
570	2.36	2.36	.6243386
600	2.34	2.34	.6190476

CONFINED AQUIFER, FULLY PENETRATING CONDITION

K = 0.5E-04 cm/sec
  = 1.2 gpd/ft2
  = 0.2E-05 ft/sec
  = 0.2 ft/day

REGRESSION COEFFICIENT = -.9867073


X AXIS:
TIME, min
Y AXIS:
LOG (H/H0)
EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.
1.0
.5
.1
.05
.01
6.2
18.6
31.0
43.4
55.8

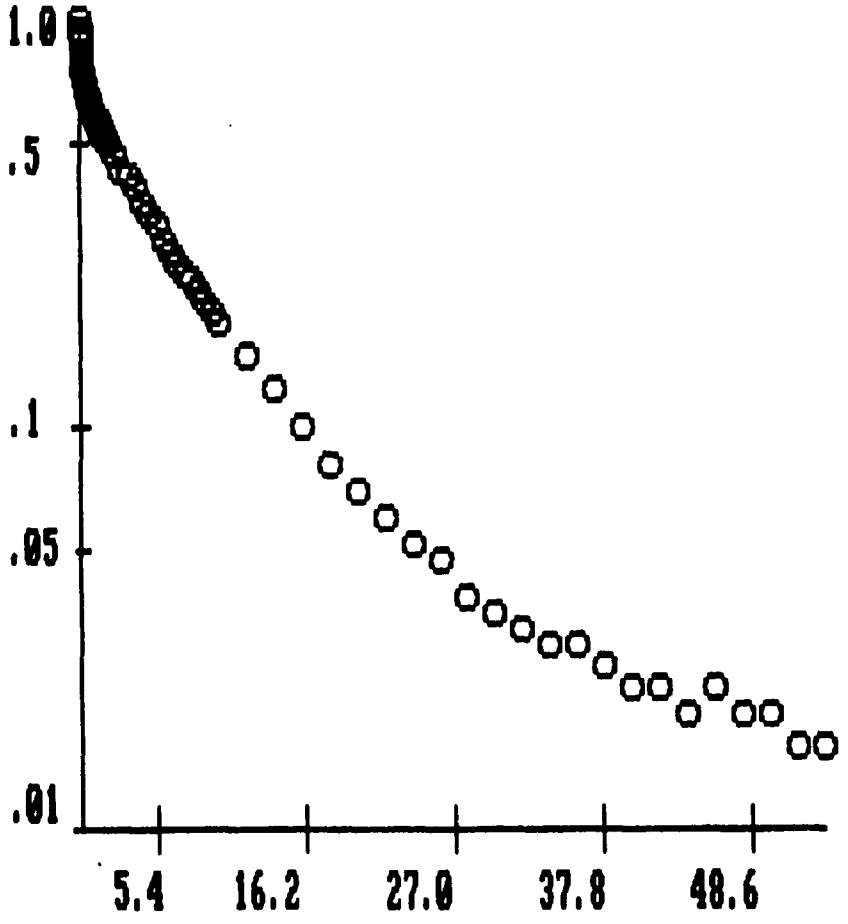
Summit National Site -- MW-15

TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
60	1.88	1.88	.5893417
64.998	1.85	1.85	.5799373
70.002	1.82	1.82	.570533
75	1.79	1.79	.5611285
79.998	1.76	1.76	.5517241
84.996	1.74	1.74	.5454545
90	1.71	1.71	.5360502
94.998	1.69	1.69	.5297806
100.002	1.66	1.66	.5203762
105	1.64	1.64	.5141066
109.998	1.62	1.62	.507837
115.002	1.6	1.60	.5015675
120	1.57	1.57	.492163
150	1.47	1.47	.4608151
180	1.37	1.37	.4294671
210	1.29	1.29	.4043887
240	1.22	1.22	.3824451
270	1.13	1.13	.354232
300	1.05	1.05	.3291536
330	.98	0.98	.30721
360	.92	0.92	.2884013
390	.86	0.86	.2695925
420	.81	0.81	.2539185
450	.76	0.76	.2382445
480	.72	0.72	.2257054
510	.68	0.68	.2131662
540	.64	0.64	.200627
570	.61	0.61	.1912226
600	.58	0.58	.1818182
720	.47	0.47	.1473354
840	.39	0.39	.1222571
960	.32	0.32	.1003135

UNCONFINED AQUIFER

K = 0.1E-03 cm/sec
= 2.3 gpd/ft2
= 0.4E-05 ft/sec
= 0.3 ft/day

REGRESSION COEFFICIENT = -.9965199



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

MW-15 (con't.)

Summit National Site -- MW-16

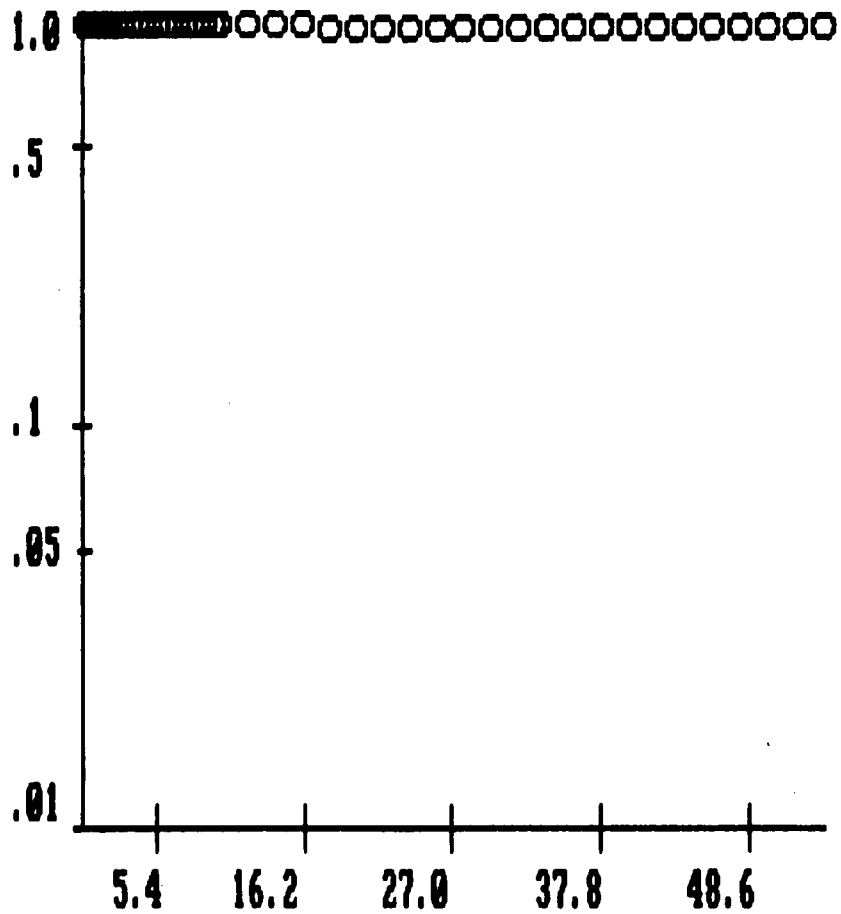
TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
4.998	4.63	4.63	1
6	4.61	4.61	.9956803
6.996	4.61	4.61	.9956803
7.998	4.61	4.61	.9956803
9	4.61	4.61	.9956803
9.996	4.61	4.61	.9956803
10.998	4.61	4.61	.9956803
12	4.61	4.61	.9956803
12.996	4.61	4.61	.9956803
13.998	4.61	4.61	.9956803
15	4.61	4.61	.9956803
15.996	4.62	4.62	.9978401
16.998	4.61	4.61	.9956803
18	4.61	4.61	.9956803
18.996	4.61	4.61	.9956803
19.998	4.61	4.61	.9956803
25.002	4.61	4.61	.9956803
30	4.62	4.62	.9978401
34.998	4.62	4.62	.9978401
40.002	4.62	4.62	.9978401
45	4.62	4.62	.9978401
49.998	4.62	4.62	.9978401
55.002	4.62	4.62	.9978401
60	4.62	4.62	.9978401
64.998	4.62	4.62	.9978401
70.002	4.62	4.62	.9978401
75	4.62	4.62	.9978401
79.998	4.62	4.62	.9978401
84.996	4.62	4.62	.9978401
90	4.62	4.62	.9978401
94.998	4.62	4.62	.9978401
100.002	4.62	4.62	.9978401
105	4.61	4.61	.9956803
109.998	4.62	4.62	.9978401
115.002	4.62	4.62	.9978401
120	4.62	4.62	.9978401
150	4.61	4.61	.9956803
180	4.61	4.61	.9956803
210	4.61	4.61	.9956803
240	4.6	4.60	.9935205
270	4.6	4.60	.9935205
300	4.6	4.60	.9935205
330	4.6	4.60	.9935205
360	4.6	4.60	.9935205
390	4.59	4.59	.9913608
420	4.59	4.59	.9913608
450	4.59	4.59	.9913608
480	4.58	4.58	.9892008
510	4.58	4.58	.9892008
540	4.58	4.58	.9892008
570	4.58	4.58	.9892008
600	4.58	4.58	.9892008
720	4.57	4.57	.9870411

840	4.56	4.56	.9848812
960	4.56	4.56	.9848812
1080	4.55	4.55	.9827214
1200	4.55	4.55	.9827214
1320	4.54	4.54	.9805616
1440	4.53	4.53	.9784018
1560	4.52	4.52	.9762419
1680	4.52	4.52	.9762419
1800	4.51	4.51	.9740821
1920	4.51	4.51	.9740821
2040	4.5	4.50	.9719222
2160	4.49	4.49	.9697624
2280	4.49	4.49	.9697624
2400	4.48	4.48	.9676026
2520	4.48	4.48	.9676026
2640	4.47	4.47	.9654427
2760	4.47	4.47	.9654427
2880	4.46	4.46	.9632829
3000	4.45	4.45	.9611231
3120	4.45	4.45	.9611231
3240	4.44	4.44	.9589632

CONFINED AQUIFER, FULLY PENETRATING CONDITION

K = 0.6E-06 cm/sec
= 0.0 gpd/ft2
= 0.2E-07 ft/sec
= 0.0 ft/day

REGRESSION COEFFICIENT = -.9939054



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

MW-16 (con't.)

Summit National Site -- MW-17

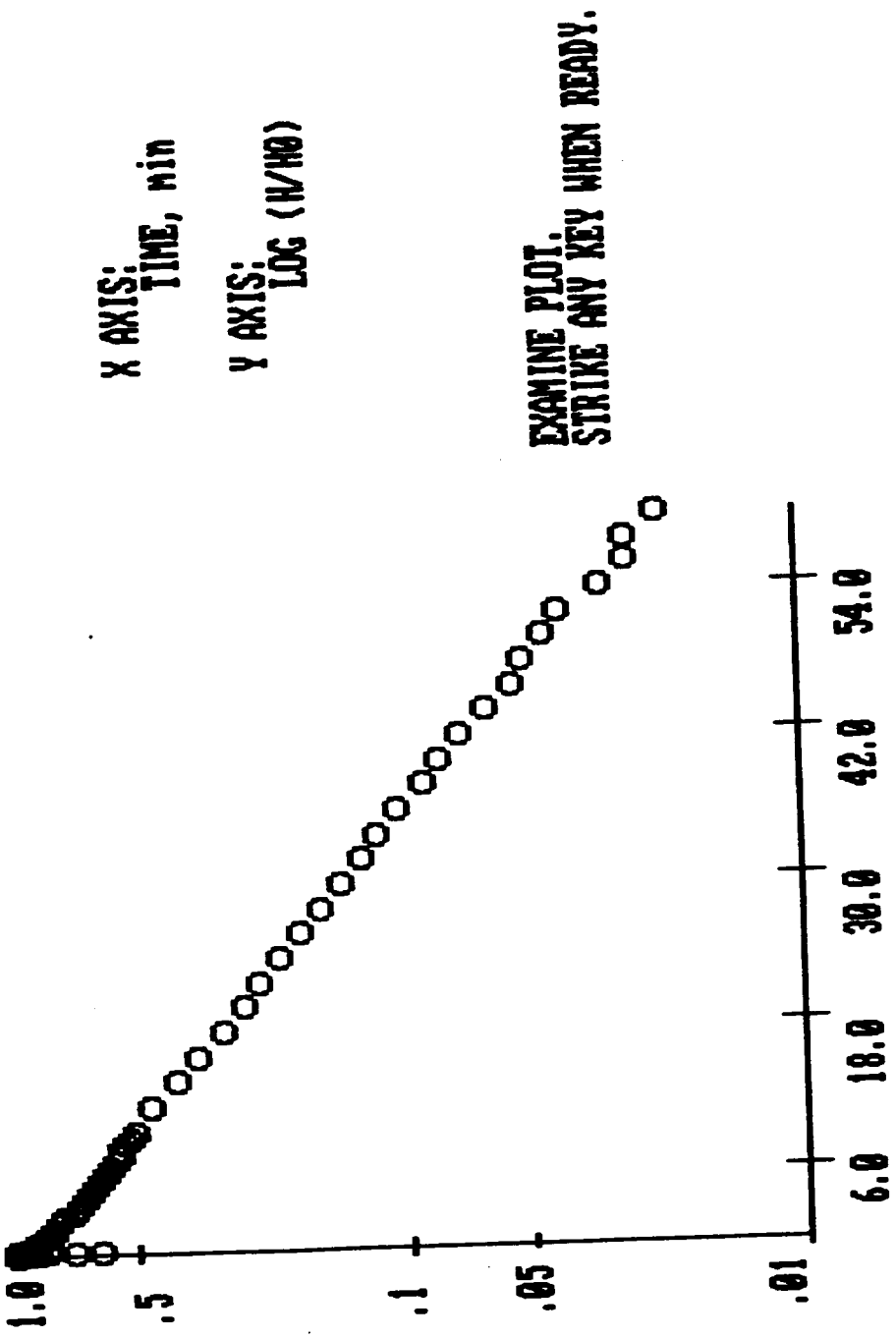
TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
30	2.11	2.11	.9336282
34.998	2.09	2.09	.9247787
40.002	2.08	2.08	.9203539
45	2.06	2.06	.9115043
49.998	2.04	2.04	.9026548
55.002	2.02	2.02	.8938053
60	2	2.00	.8849558
64.998	1.98	1.98	.8761062
70.002	1.96	1.96	.8672566
75	1.95	1.95	.8628318
79.998	1.93	1.93	.8539822
84.996	1.91	1.91	.8451327
90	1.9	1.90	.8407079
94.998	1.89	1.89	.8362831
100.002	1.87	1.87	.8274336
105	1.86	1.86	.8230089
109.998	1.85	1.85	.8185841
115.002	1.83	1.83	.8097345
120	1.82	1.82	.8053097
150	1.75	1.75	.7743363
180	1.69	1.69	.7477876
210	1.63	1.63	.721239
240	1.58	1.58	.6991151
270	1.52	1.52	.6725664
300	1.48	1.48	.6548673
330	1.43	1.43	.6327433
360	1.39	1.39	.6150443
390	1.35	1.35	.5973451
420	1.31	1.31	.579646
450	1.28	1.28	.5663717
480	1.24	1.24	.5486726
510	1.23	1.23	.5442478
540	1.18	1.18	.5221239
570	1.15	1.15	.5088495
600	1.12	1.12	.4955752
720	1.02	1.02	.4513274
840	.88	0.88	.3893805
960	.76	0.76	.3362832
1080	.67	0.67	.2964602
1200	.59	0.59	.2610619
1320	.53	0.53	.2345133
1440	.47	0.47	.2079646
1560	.42	0.42	.1858407
1680	.37	0.37	.1637168
1800	.33	0.33	.1460177
1920	.29	0.29	.1283186
2040	.26	0.26	.1150442

2160	.23	0.23	.1017699
2280	.2	0.20	8.849558E-02
2400	.18	0.18	7.964602E-02
2520	.16	0.16	7.079646E-02
2640	.14	0.14	.0619469
2760	.12	0.12	5.309734E-02
2880	.11	0.11	4.867257E-02
3000	.1	0.10	4.424779E-02
3120	9.000001E-02		
		0.09	3.982301E-02
3240	.07	0.07	3.097345E-02
3360	.06	0.06	2.654867E-02
3480	.06	0.06	2.654867E-02
3600	.05	0.05	2.212389E-02

UNCONFINED AQUIFER

K = 0.6E-04 cm/sec
= 1.2 gpd/ft2
= 0.2E-05 ft/sec
= 0.2 ft/day

REGRESSION COEFFICIENT = -.9997093


X AXIS:
TIME, min
Y AXIS:
LOG (H/H0)
EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.
1.0
.5
.1
.05
.01
6.0
18.0
30.0
42.0
54.0

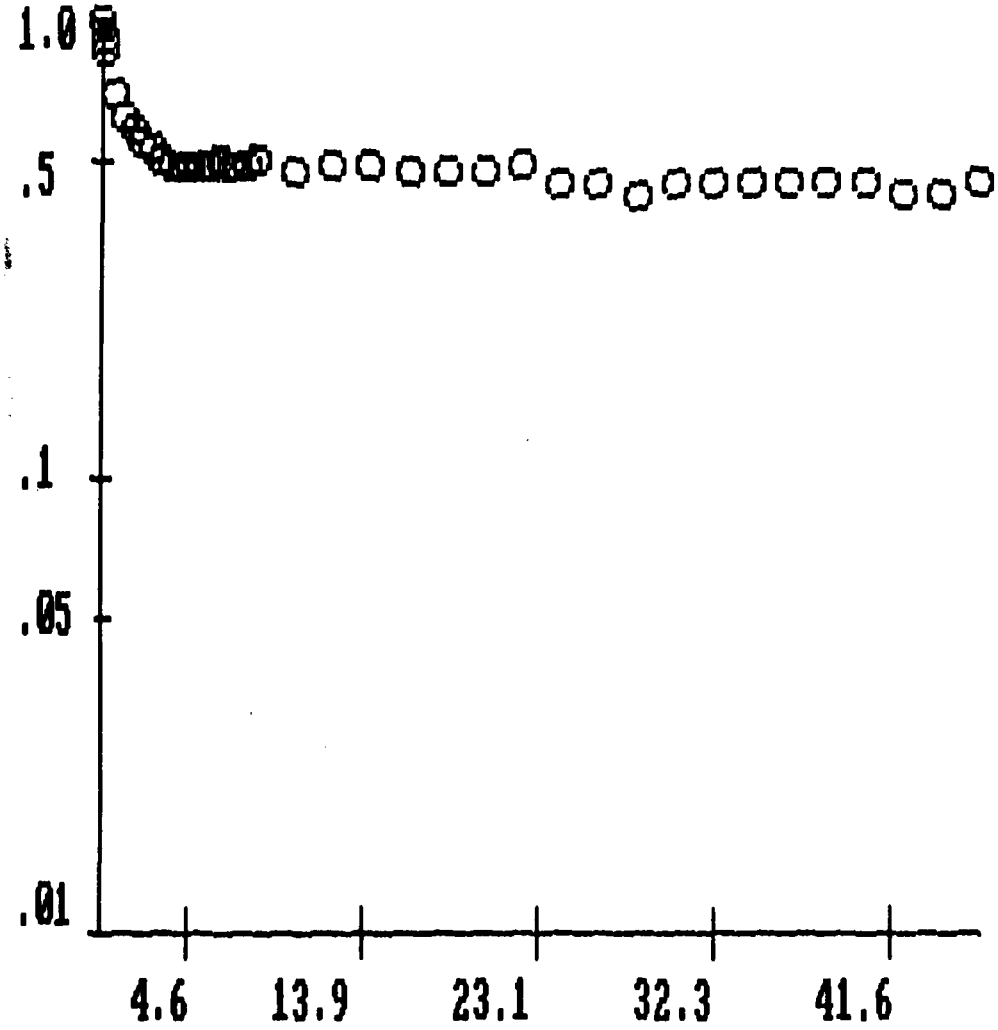
[illegible] National Site -- MW-1S

TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
10.002	.41	0.41	.8541667
40.002	.33	0.33	.6875001
70.002	.29	0.29	.6041666
100.002	.28	0.28	.5833334
130.002	.26	0.26	.5416666
160.002	.25	0.25	.5208334
190.002	.24	0.24	.5
220.002	.23	0.23	.4791667

UNCONFINED AQUIFER

K = 0.8E-04 cm/sec
= 1.7 gpd/ft2
= 0.3E-05 ft/sec
= 0.2 ft/day

REGRESSION COEFFICIENT = -.9423798



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

MM-18 (cont'd.)

1LIST 2RUN← 3LOAD" 4SAVE" 5CONT← 6,"LPT1 7TRON← 8TROFF← 9KEY 0SCREEN

Plume National Site -- MW-13

TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
3	3.16	3.16	1
3.996	2.88	2.88	.9113924
4.998	2.75	2.75	.8702532
6	2.62	2.62	.8291139
6.996	2.5	2.50	.7911393
7.998	2.38	2.38	.7531646
9	2.26	2.26	.7151899
9.996	2.16	2.16	.6835443
10.998	2.06	2.06	.6518987
12	1.97	1.97	.6234177
12.996	1.88	1.88	.5949368
13.998	1.79	1.79	.5664557
15	1.71	1.71	.5411393
15.996	1.64	1.64	.5189873
16.998	1.57	1.57	.4968355
18	1.51	1.51	.4778481
18.996	1.46	1.46	.4620253
19.998	1.41	1.41	.4462025
25.002	1.21	1.21	.3829114
30	1.08	1.08	.3417722
34.998	.98	0.98	.3101266
40.002	.9	0.90	.2848101
45	.83	0.83	.2626582
49.998	.77	0.77	.2436709
55.002	.71	0.71	.2246836
60	.66	0.66	.2089873
64.998	.61	0.61	.193038
70.002	.57	0.57	.1803798
75	.53	0.53	.1677215
79.998	.5	0.50	.1582279
84.996	.47	0.47	.1487342
90	.45	0.45	.1424051
94.998	.43	0.43	.1360759
100.002	.4	0.40	.1265823
105	.39	0.39	.1234177
109.998	.37	0.37	.1170886
115.002	.36	0.36	.1139241
120	.34	0.34	.1075949

UNCONFINED AQUIFER

K = 0.6E-03 cm/sec
= 13.3 gpd/ft2
= 0.2E-04 ft/sec
= 1.8 ft/day

REGRESSION COEFFICIENT = -.9713761

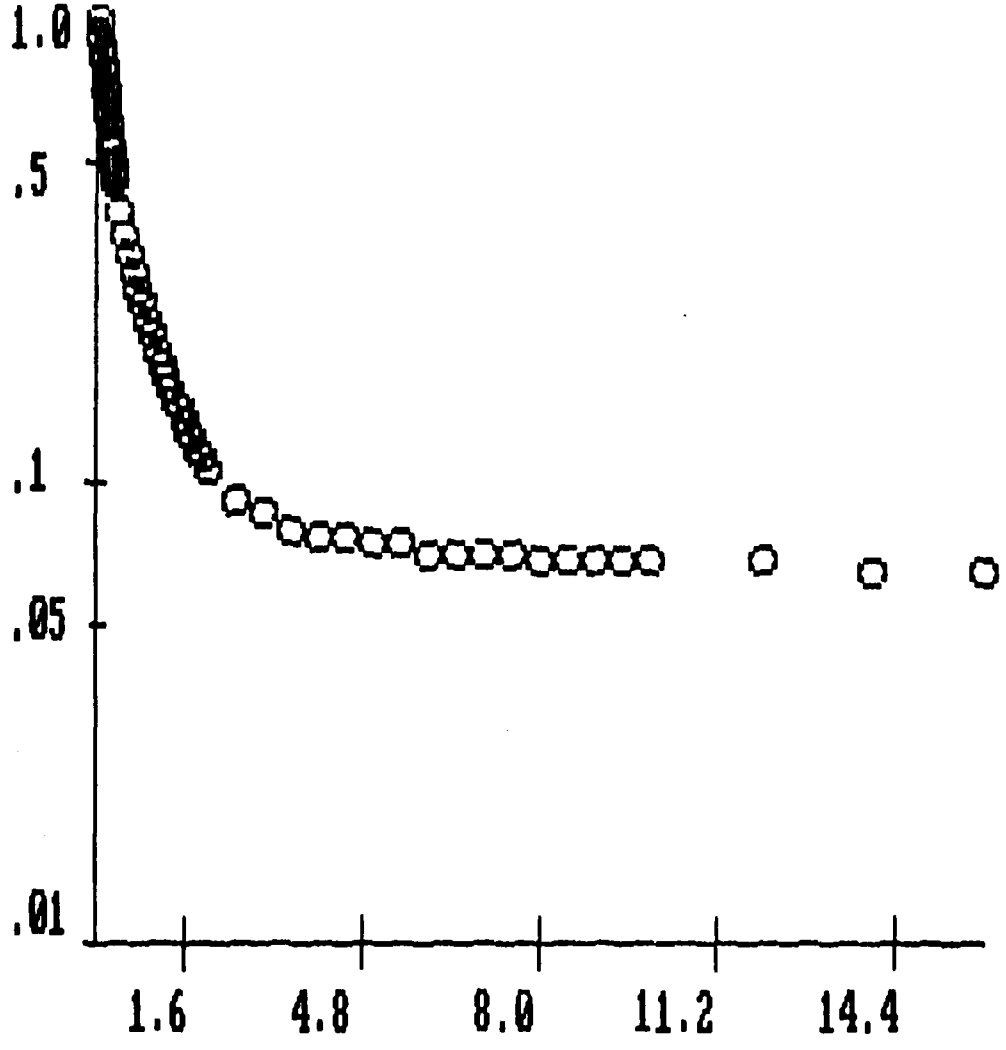
E. Crit National Site -- MW-19

TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/HO
19.998	1.41	1.41	.4463025
25.002	1.21	1.21	.3829114
30	1.08	1.08	.3417722
34.998	.98	0.98	.3101266
40.002	.9	0.90	.2848101
45	.83	0.83	.2626582
49.998	.77	0.77	.2436709
55.002	.71	0.71	.2246836
60	.66	0.66	.2088608
64.998	.61	0.61	.193038
70.002	.57	0.57	.1803798
75	.53	0.53	.1677215
79.998	.5	0.50	.1582279
84.996	.47	0.47	.1487342
90	.45	0.45	.1424051
94.998	.43	0.43	.1360759
100.002	.4	0.40	.1265823
105	.39	0.39	.1234177
109.998	.37	0.37	.1170886
115.002	.36	0.36	.1139241
120	.34	0.34	.1075949

UNCONFINED AQUIFER

K = 0.5E-03 cm/sec
= 9.8 gpd/ft2
= 0.65-04 ft/sec
= 1.3 ft/day

REGRESSION COEFFICIENT = -.9887367



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

YW-19 (con't.)

1LIST 2RUN← 3LOAD" 4SAVE" 5CONT← 6,"LPT1 7TRON← 8TROFF← 9KEY 0SCREEN

SUMMIT NATIONAL SITE -- MW-20

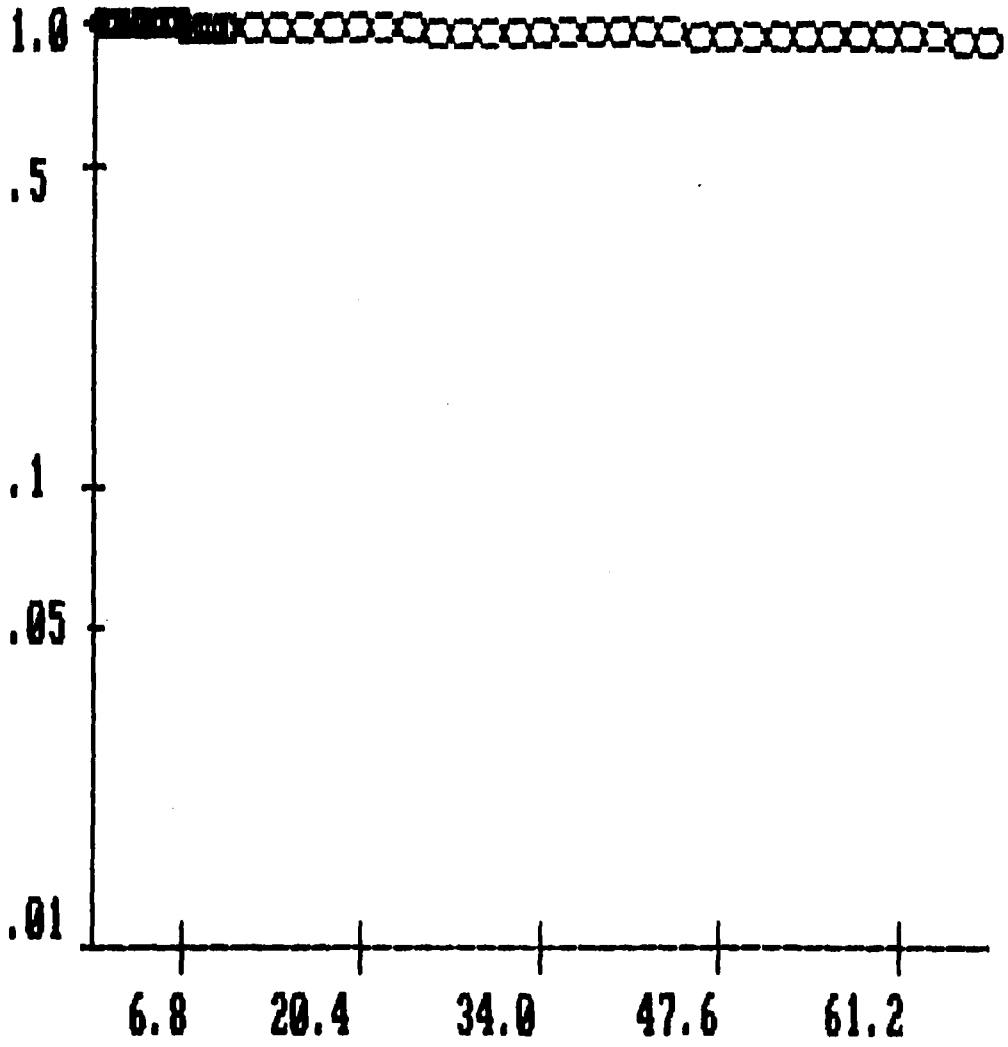
TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	S/H0
40.002	4.4	4.40	1
45	4.4	4.40	1
49.998	4.4	4.40	1
55.002	4.39	4.39	.9977272
60	4.39	4.39	.9977272
64.998	4.39	4.39	.9977272
70.002	4.39	4.39	.9977272
75	4.39	4.39	.9977272
79.998	4.39	4.39	.9977272
84.996	4.39	4.39	.9977272
90	4.39	4.39	.9977272
94.998	4.39	4.39	.9977272
100.002	4.39	4.39	.9977272
105	4.38	4.38	.9954546
109.998	4.38	4.38	.9954546
115.002	4.38	4.38	.9954546
120	4.38	4.38	.9954546
150	4.38	4.38	.9954546
180	4.37	4.37	.9931818
210	4.36	4.36	.9909091
240	4.36	4.36	.9909091
270	4.36	4.36	.9909091
300	4.35	4.35	.9886363
330	4.35	4.35	.9886363
360	4.34	4.34	.9863637
390	4.34	4.34	.9863637
420	4.33	4.33	.9840909
450	4.33	4.33	.9840909
480	4.33	4.33	.9840909
510	4.32	4.32	.9818182
540	4.32	4.32	.9818182
570	4.31	4.31	.9795455
600	4.31	4.31	.9795455
720	4.29	4.29	.975
840	4.28	4.28	.9727274
960	4.26	4.26	.9681819
1080	4.25	4.25	.9659091
1200	4.23	4.23	.9613637
1320	4.22	4.22	.9590909
1440	4.21	4.21	.9568182
1560	4.19	4.19	.9522727
1680	4.18	4.18	.9499999
1800	4.17	4.17	.9477272
1920	4.15	4.15	.9431818
2040	4.14	4.14	.940909
2160	4.13	4.13	.9386364
2280	4.11	4.11	.9340909
2400	4.1	4.10	.9318182

3520	4.09	4.09	.9295455
3640	4.08	4.08	.9272727
3760	4.07	4.07	.9250001
3880	4.05	4.05	.9204546
3000	4.04	4.04	.9181818
3120	4.03	4.03	.9159092
3240	4.02	4.02	.9136364
3360	4	4.00	.9090909
3480	3.99	3.99	.9068181
3600	3.98	3.98	.9045455
3720	3.97	3.97	.9022728
3840	3.96	3.96	.9
3960	3.95	3.95	.8977273
4080	3.94	3.94	.8954545

CONFINED AQUIFER, FULLY PENETRATING CONDITION

κ = 0.9E-05 cm/sec
= 0.2 gpd/ft2
= 0.3E-06 ft/sec
= 0.0 ft/day

REGRESSION COEFFICIENT = -.9981674



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

MW-20 (con't.)

SUMMIT NATIONAL SITE -- MW-21

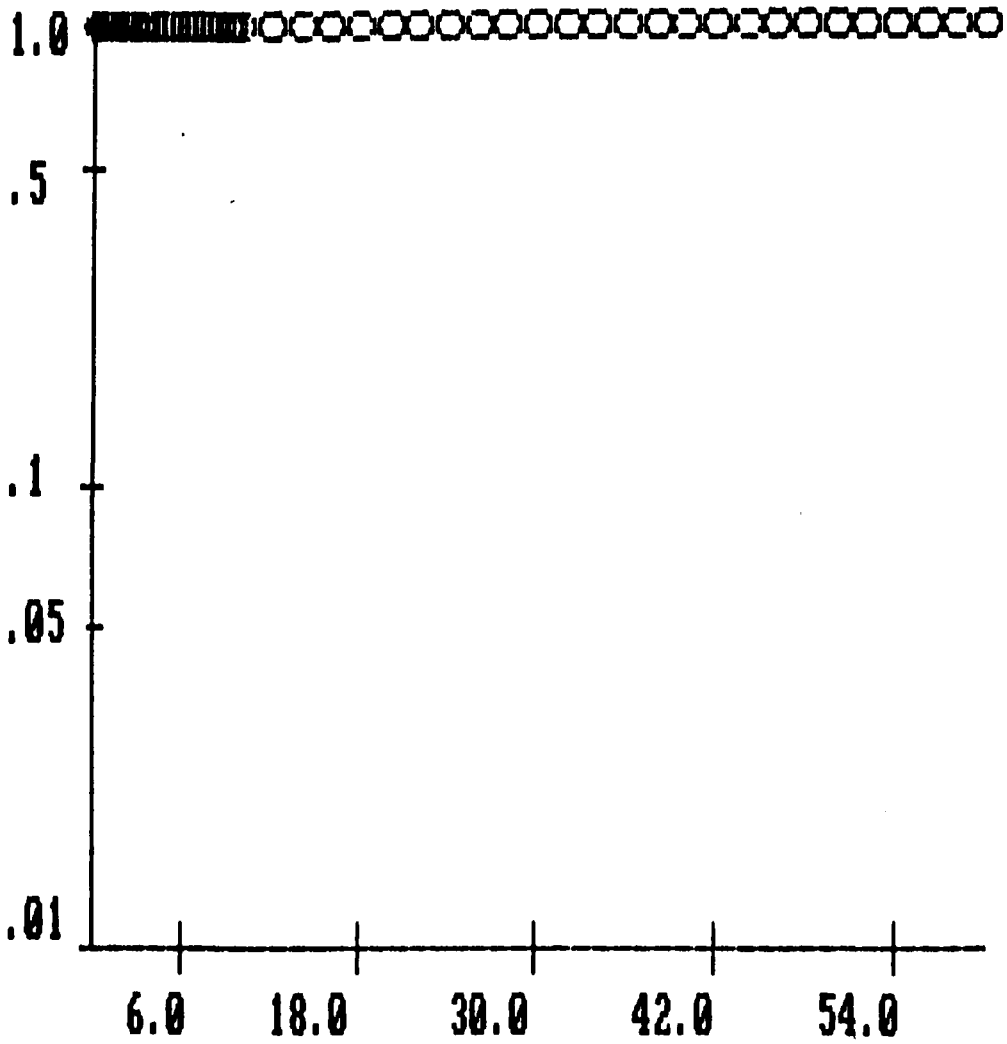
TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
30	3.61	3.61	1
34.998	3.61	3.61	1
40.002	3.61	3.61	1
45	3.61	3.61	1
49.998	3.61	3.61	1
55.002	3.61	3.61	1
60	3.61	3.61	1
64.998	3.61	3.61	1
70.002	3.61	3.61	1
75	3.61	3.61	1
79.998	3.61	3.61	1
84.996	3.61	3.61	1
90	3.61	3.61	1
94.998	3.61	3.61	1
100.002	3.61	3.61	1
105	3.61	3.61	1
109.998	3.61	3.61	1
115.002	3.61	3.61	1
120	3.61	3.61	1
150	3.61	3.61	1
180	3.61	3.61	1
210	3.61	3.61	1
240	3.61	3.61	1
270	3.61	3.61	1
300	3.61	3.61	1
330	3.61	3.61	1
360	3.61	3.61	1
390	3.61	3.61	1
420	3.61	3.61	1
450	3.61	3.61	1
480	3.61	3.61	1
510	3.61	3.61	1
540	3.61	3.61	1
570	3.61	3.61	1
600	3.61	3.61	1
720	3.61	3.61	1
840	3.61	3.61	1
960	3.61	3.61	1
1080	3.61	3.61	1
1200	3.61	3.61	1
1320	3.61	3.61	1
1440	3.61	3.61	1
1560	3.61	3.61	1
1680	3.61	3.61	1
1800	3.61	3.61	1
1920	3.61	3.61	1
2040	3.61	3.61	1
2160	3.61	3.61	1

2280	3.61	3.61	1
2400	3.61	3.61	1
2520	3.61	3.61	1
2640	3.6	3.60	.99723
2760	3.61	3.61	1
2880	3.61	3.61	1
3000	3.61	3.61	1
3120	3.61	3.61	1
3240	3.61	3.61	1
3360	3.61	3.61	1
3480	3.61	3.61	1
3600	3.61	3.61	1

CONFINED AQUIFER, PARTIALLY PENETRATING CONDITION

K = 0.2E-07 cm/sec
= 0.0 gpd/ft2
= 0.5E-09 ft/sec
= 0.0 ft/day

REGRESSION COEFFICIENT = -.1880114



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

SUMMIT NATIONAL SITE -- MW-22

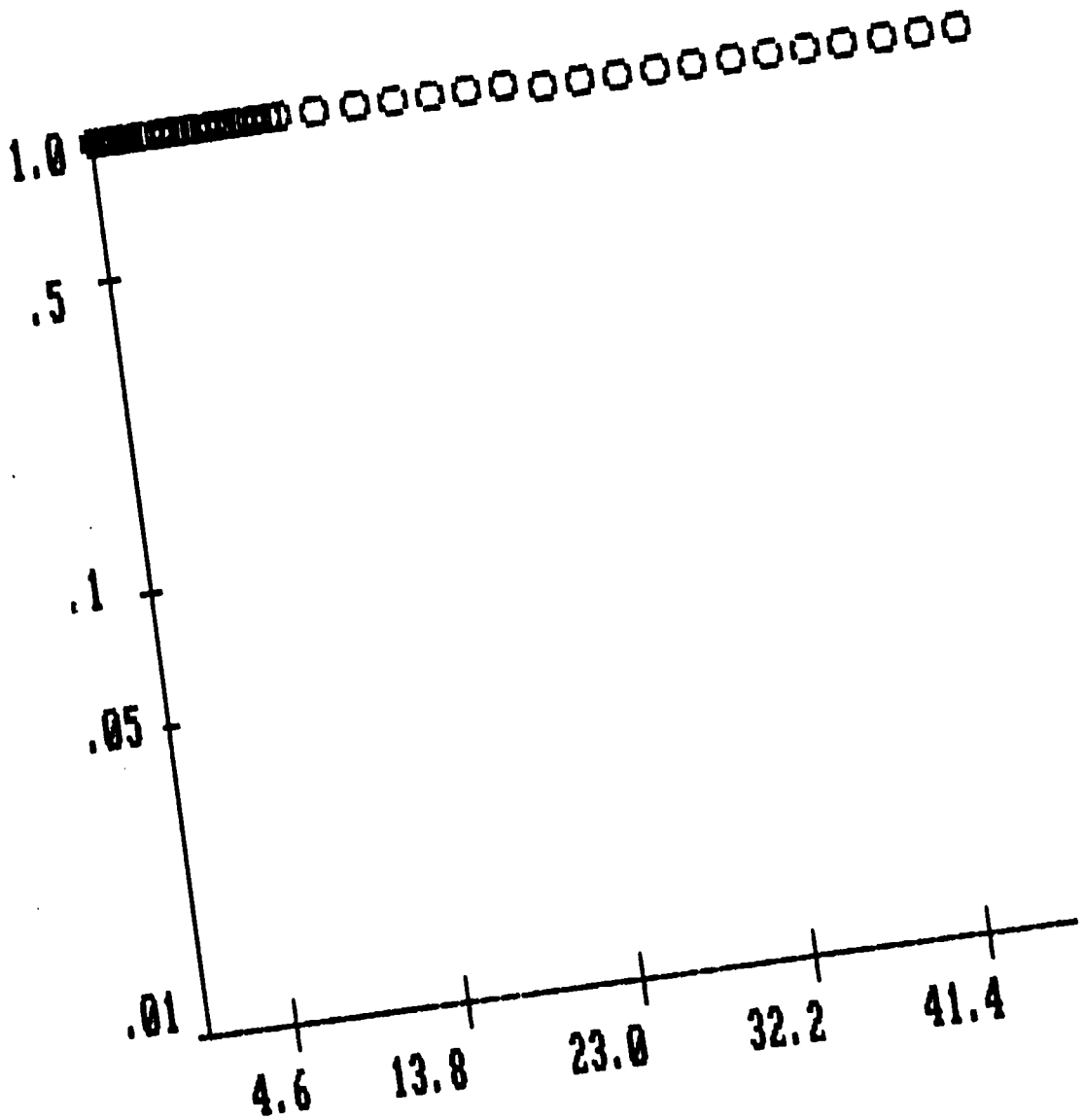
TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/HO
9.996	3.66	3.66	.9972752
10.998	3.66	3.66	.9972752
12	3.66	3.66	.9972752
12.996	3.66	3.66	.9972752
13.998	3.67	3.67	1
15	3.67	3.67	1
15.996	3.67	3.67	1
16.998	3.67	3.67	1
18	3.67	3.67	1
18.996	3.67	3.67	1
19.998	3.67	3.67	1
25.002	3.67	3.67	1
30	3.67	3.67	1
34.998	3.67	3.67	1
40.002	3.67	3.67	1
45	3.66	3.66	.9972752
49.998	3.66	3.66	.9972752
55.002	3.66	3.66	.9972752
60	3.66	3.66	.9972752
64.998	3.66	3.66	.9972752
70.002	3.66	3.66	.9972752
75	3.66	3.66	.9972752
79.998	3.66	3.66	.9972752
84.996	3.66	3.66	.9972752
90	3.66	3.66	.9972752
94.998	3.66	3.66	.9972752
100.002	3.66	3.66	.9972752
105	3.66	3.66	.9972752
109.998	3.66	3.66	.9972752
115.002	3.66	3.66	.9972752
120	3.66	3.66	.9972752
150	3.66	3.66	.9972752
180	3.66	3.66	.9972752
210	3.66	3.66	.9972752
240	3.66	3.66	.9972752
270	3.65	3.65	.9945504
300	3.65	3.65	.9945504
330	3.65	3.65	.9945504
360	3.65	3.65	.9945504
390	3.65	3.65	.9945504
420	3.65	3.65	.9945504
450	3.65	3.65	.9945504
480	3.65	3.65	.9945504
510	3.65	3.65	.9945504
540	3.65	3.65	.9945504
570	3.65	3.65	.9945504
600	3.64	3.64	.9918256
720	3.64	3.64	.9918256

840	3.64	3.64	.9918856
960	3.63	3.63	.9891009
1080	3.63	3.63	.9891009
1200	3.63	3.63	.9891009
1320	3.62	3.62	.9863759
1440	3.61	3.61	.9836512
1560	3.6	3.60	.9809264
1680	3.6	3.60	.9809264
1800	3.6	3.60	.9809264
1920	3.59	3.59	.9782016
2040	3.59	3.59	.9782016
2160	3.59	3.59	.9782016
2280	3.58	3.58	.9754769
2400	3.58	3.58	.9754769
2520	3.57	3.57	.9727521
2640	3.57	3.57	.9727521
2760	3.56	3.56	.9700272

CONFINED AQUIFER, FULLY PENETRATING CONDITION

K = 0.3E-05 cm/sec
= 0.1 gpd/ft2
= 0.1E-06 ft/sec
= 0.0 ft/day

REGRESSION COEFFICIENT = -.9914401



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

MW-22 (con't.)

SUMMIT NATIONAL SITE -- MW-23

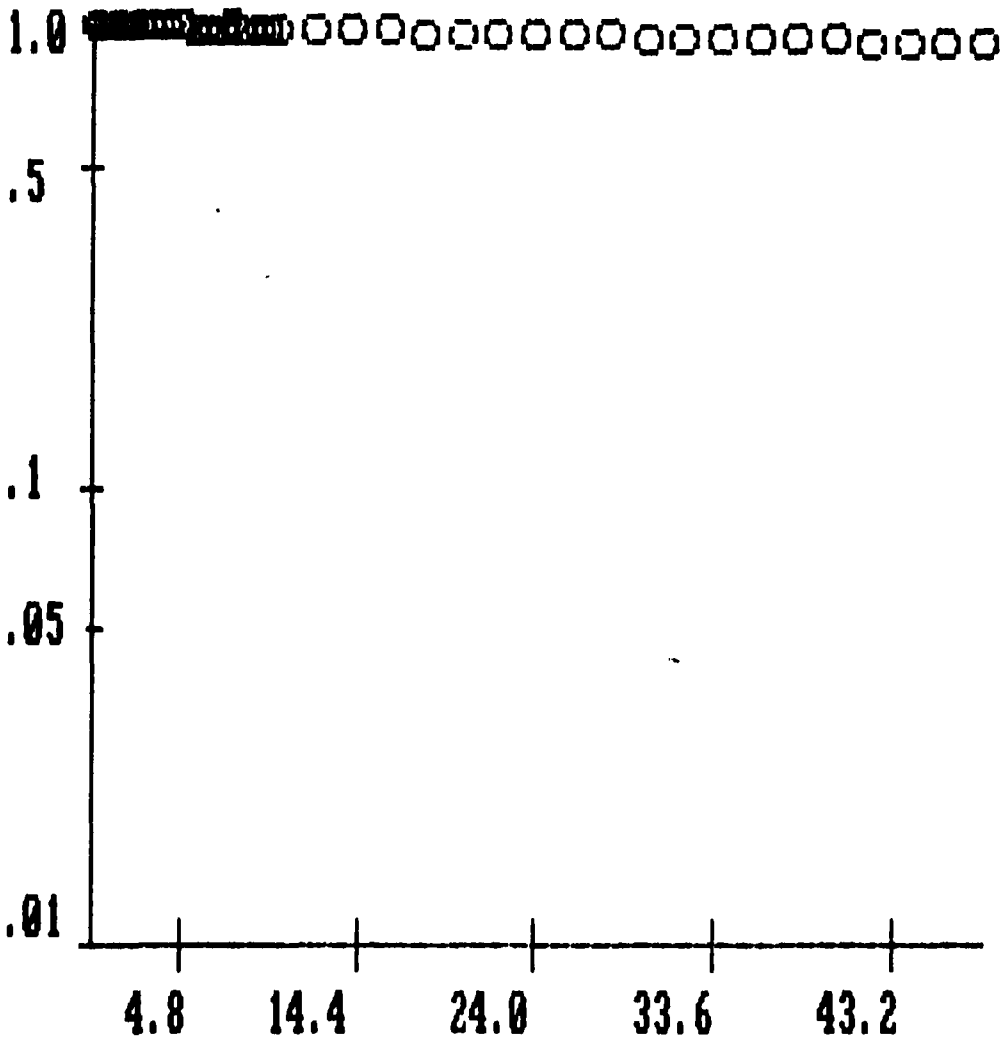
TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
6.996	3.65	3.65	1
7.998	3.65	3.65	1
9	3.64	3.64	.9972603
9.996	3.64	3.64	.9972603
10.998	3.64	3.64	.9972603
12	3.64	3.64	.9972603
12.996	3.64	3.64	.9972603
13.998	3.64	3.64	.9972603
15	3.64	3.64	.9972603
15.996	3.64	3.64	.9972603
16.998	3.64	3.64	.9972603
18	3.64	3.64	.9972603
18.996	3.64	3.64	.9972603
19.998	3.64	3.64	.9972603
25.002	3.64	3.64	.9972603
30	3.64	3.64	.9972603
34.998	3.64	3.64	.9972603
40.002	3.64	3.64	.9972603
45	3.64	3.64	.9972603
49.998	3.64	3.64	.9972603
55.002	3.64	3.64	.9972603
60	3.63	3.63	.9945207
64.998	3.64	3.64	.9972603
70.002	3.64	3.64	.9972603
75	3.64	3.64	.9972603
79.998	3.63	3.63	.9945207
84.996	3.63	3.63	.9945207
90	3.63	3.63	.9945207
94.998	3.64	3.64	.9972603
100.002	3.63	3.63	.9945207
105	3.63	3.63	.9945207
109.998	3.63	3.63	.9945207
115.002	3.63	3.63	.9945207
120	3.63	3.63	.9945207
150	3.62	3.62	.9917808
180	3.62	3.62	.9917808
210	3.62	3.62	.9917808
240	3.61	3.61	.9890411
270	3.6	3.60	.9863014
300	3.6	3.60	.9863014
330	3.59	3.59	.9835616
360	3.59	3.59	.9835616
390	3.59	3.59	.9835616
420	3.58	3.58	.9808219
450	3.6	3.60	.9863014
480	3.58	3.58	.9808219
510	3.58	3.58	.9808219
540	3.57	3.57	.9780822

570	3.57	3.57	.9780822
600	3.56	3.56	.9753425
720	3.53	3.53	.9671232
840	3.52	3.52	.9643836
960	3.5	3.50	.9589041
1080	3.48	3.48	.9534247
1200	3.46	3.46	.9479452
1320	3.45	3.45	.9452055
1440	3.43	3.43	.9397261
1560	3.41	3.41	.9342466
1680	3.39	3.39	.9287671
1800	3.37	3.37	.9232876
1920	3.36	3.36	.9205479
2040	3.34	3.34	.9150684
2160	3.33	3.33	.9123288
2280	3.33	3.33	.9123288
2400	3.3	3.30	.9041096
2520	3.27	3.27	.8958904
2640	3.26	3.26	.8931507
2760	3.24	3.24	.8876712
2880	3.22	3.22	.8821918

CONFINED AQUIFER, FULLY PENETRATING CONDITION

K = 0.1E-04 cm/sec
= 0.3 gpd/ft2
= 0.4E-06 ft/sec
= 0.0 ft/day

REGRESSION COEFFICIENT = -.9989186



X AXIS:
TIME, min

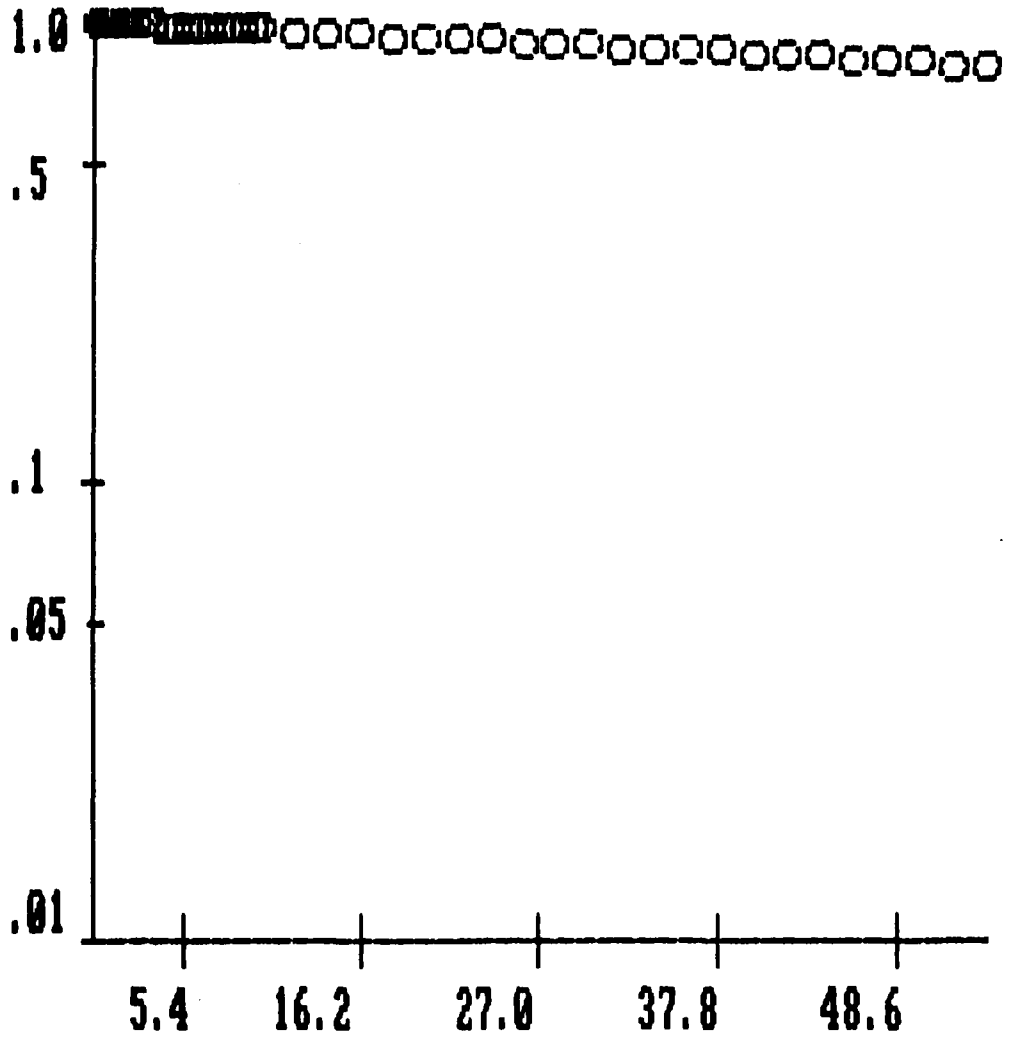
Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

MW-33 (con't.)

SUMMIT NATIONAL SITE -- MW-24

TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/HO
6	3.67	3.67	1
6.996	3.67	3.67	1
7.998	3.67	3.67	1
9	3.67	3.67	1
9.996	3.67	3.67	1
10.998	3.67	3.67	1
12	3.67	3.67	1
12.996	3.67	3.67	1
13.998	3.67	3.67	1
15	3.67	3.67	1
15.996	3.67	3.67	1
16.998	3.67	3.67	1
18	3.67	3.67	1
18.996	3.67	3.67	1
19.998	3.67	3.67	1
25.002	3.67	3.67	1
30	3.67	3.67	1
34.998	3.67	3.67	1
40.002	3.67	3.67	1
45	3.67	3.67	1
49.998	3.67	3.67	1
55.002	3.66	3.66	.9972752
60	3.66	3.66	.9972752
64.998	3.66	3.66	.9972752
70.002	3.66	3.66	.9972752
75	3.66	3.66	.9972752
79.998	3.66	3.66	.9972752
84.996	3.65	3.65	.9945504
90	3.65	3.65	.9945504
94.998	3.65	3.65	.9945504
100.002	3.65	3.65	.9945504
105	3.65	3.65	.9945504
109.998	3.65	3.65	.9945504
115.002	3.65	3.65	.9945504
120	3.65	3.65	.9945504
150	3.64	3.64	.9918256
180	3.63	3.63	.9891009
210	3.62	3.62	.9863759
240	3.61	3.61	.9836512
270	3.61	3.61	.9836512
300	3.6	3.60	.9809264
330	3.59	3.59	.9782016
360	3.58	3.58	.9754769
390	3.58	3.58	.9754769
420	3.57	3.57	.9727521
450	3.56	3.56	.9700272
480	3.55	3.55	.9673024
510	3.54	3.54	.9645777



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

MW-24 (con't.)

SUMMIT NATIONAL SITE -- MW-25

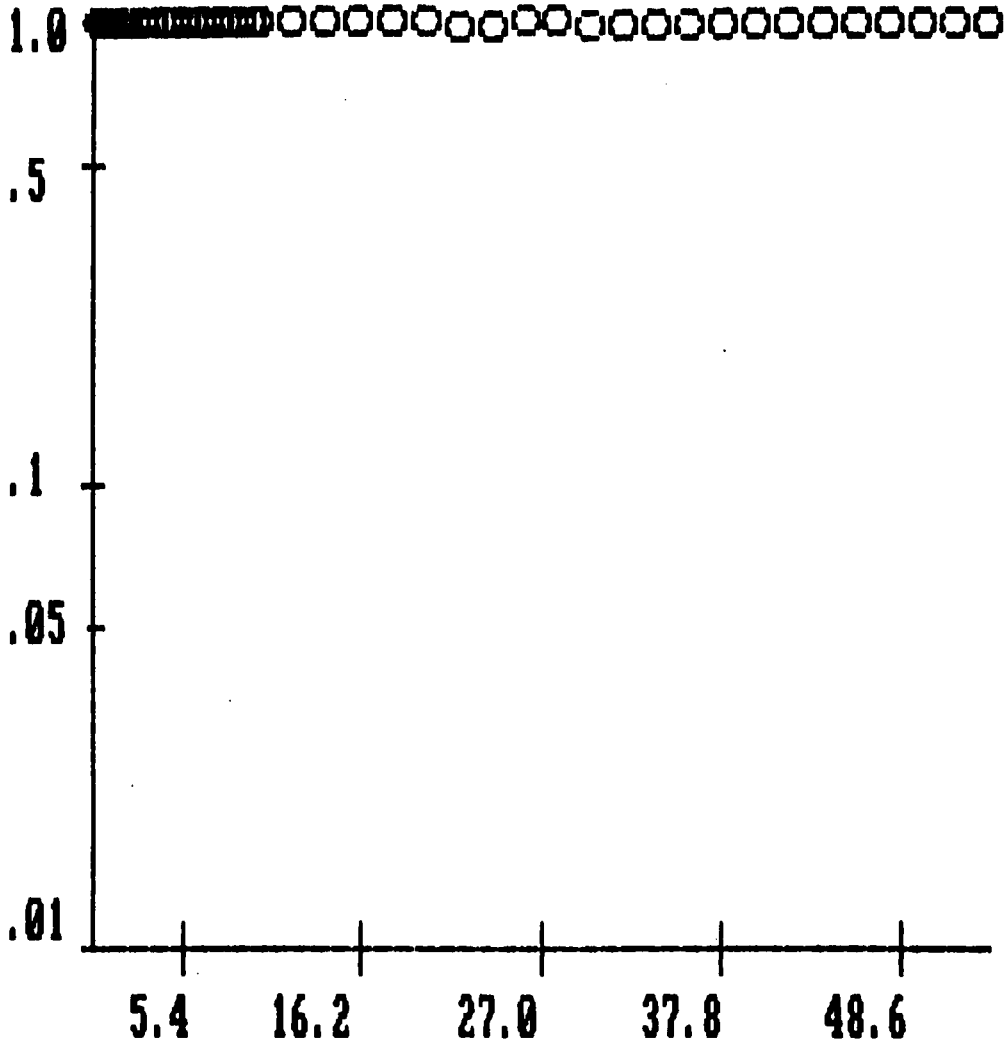
TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
18	3.6	3.60	1
18.996	3.6	3.60	1
19.998	3.6	3.60	1
25.002	3.6	3.60	1
30	3.6	3.60	1
34.998	3.6	3.60	1
40.002	3.6	3.60	1
45	3.6	3.60	1
49.998	3.6	3.60	1
55.002	3.6	3.60	1
60	3.6	3.60	1
64.998	3.6	3.60	1
70.002	3.6	3.60	1
75	3.6	3.60	1
79.998	3.6	3.60	1
84.996	3.6	3.60	1
90	3.59	3.59	.9972222
94.998	3.59	3.59	.9972222
100.002	3.6	3.60	1
105	3.6	3.60	1
109.998	3.6	3.60	1
115.002	3.6	3.60	1
120	3.59	3.59	.9972222
150	3.6	3.60	1
180	3.59	3.59	.9972222
210	3.59	3.59	.9972222
240	3.59	3.59	.9972222
270	3.59	3.59	.9972222
300	3.58	3.58	.9944444
330	3.58	3.58	.9944444
360	3.58	3.58	.9944444
390	3.58	3.58	.9944444
420	3.58	3.58	.9944444
450	3.58	3.58	.9944444
480	3.57	3.57	.9916666
510	3.57	3.57	.9916666
540	3.57	3.57	.9916666
570	3.57	3.57	.9916666
600	3.57	3.57	.9916666
720	3.58	3.58	.9944444
840	3.58	3.58	.9944444
960	3.55	3.55	.9861111
1080	3.56	3.56	.9888889
1200	3.55	3.55	.9861111
1320	3.54	3.54	.9833333
1440	3.54	3.54	.9833333
1560	3.55	3.55	.9861111
1680	3.55	3.55	.9861111

1800	3.54	3.54	.9833333
1920	3.53	3.53	.9805555
2040	3.53	3.53	.9805555
2160	3.53	3.53	.9805555
2280	3.52	3.52	.9777778
2400	3.52	3.52	.9777778
2520	3.52	3.52	.9777778
2640	3.51	3.51	.975
2760	3.51	3.51	.975
2880	3.51	3.51	.975
3000	3.5	3.50	.9722222
3120	3.5	3.50	.9722222
3240	3.5	3.50	.9722222

CONFINED AQUIFER, NEARLY FULL PENETRATION WITH EQUATION FOR FULL PENETRATION
D

K = 0.5E-05 cm/sec
= 0.1 gpd/ft2
= 0.1E-06 ft/sec
= 0.0 ft/day

REGRESSION COEFFICIENT = -.9827466



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

MW-25 (con't.)

SUMMIT NATIONAL SITE -- MW-26

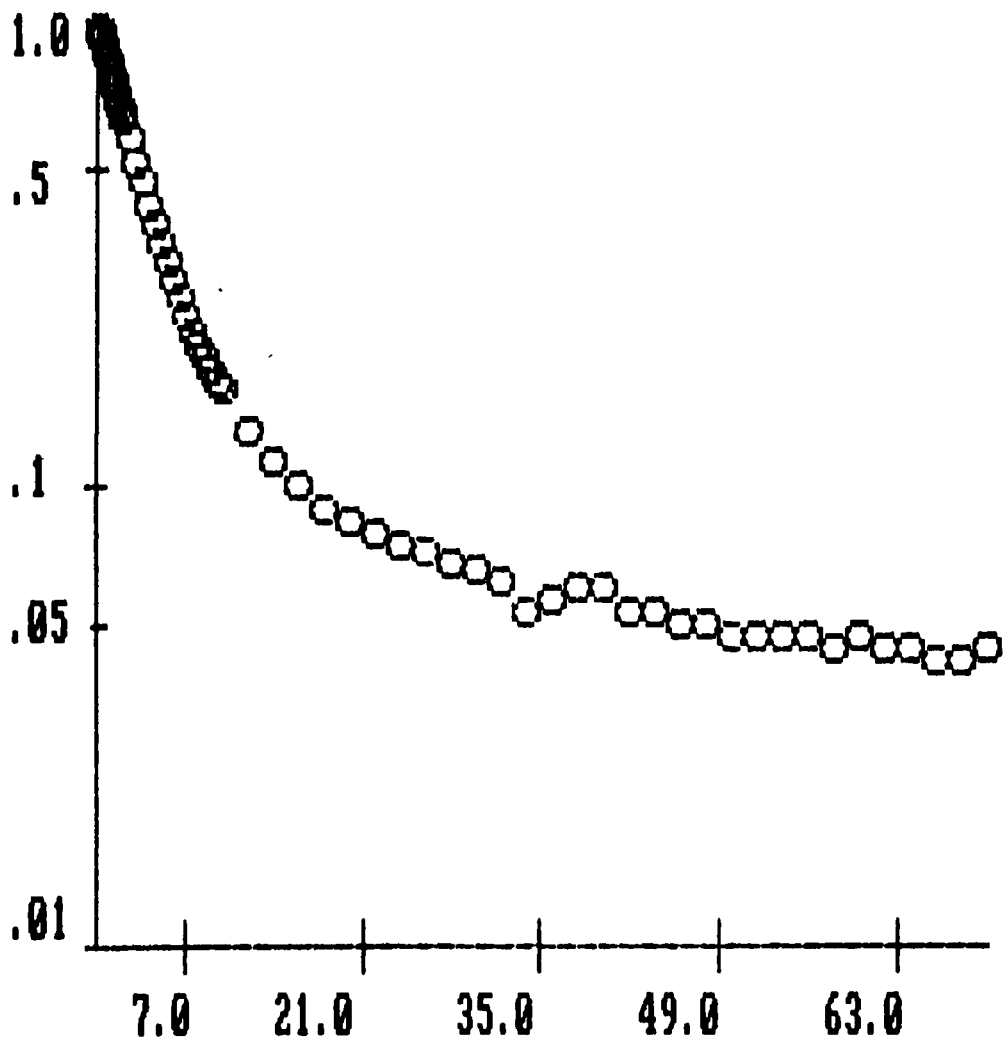
TIME (seconds)	WATER LEVEL (feet)	DRAWDOWN (feet)	H/H0
6.996	3.41	3.41	1
7.998	3.35	3.35	.9824046
9	3.36	3.36	.9853372
9.996	3.38	3.38	.9912024
10.998	3.36	3.36	.9853372
12	3.37	3.37	.9882697
12.996	3.37	3.37	.9882697
13.998	3.37	3.37	.9882697
15	3.33	3.33	.9765396
15.996	3.33	3.33	.9765396
16.998	3.35	3.35	.9824046
18	3.32	3.32	.9736069
18.996	3.3	3.30	.9677419
19.998	3.29	3.29	.9648094
25.002	3.21	3.21	.941349
30	3.14	3.14	.9208211
34.998	3.08	3.08	.9032258
40.002	3.01	3.01	.8826979
45	2.95	2.95	.8651026
49.998	2.89	2.89	.8475073
55.002	2.83	2.83	.829912
60	2.77	2.77	.8123166
64.998	2.72	2.72	.797654
70.002	2.66	2.66	.7800586
75	2.61	2.61	.7653959
79.998	2.56	2.56	.7507331
84.996	2.51	2.51	.7360704
90	2.46	2.46	.7214076
94.998	2.41	2.41	.7067449
100.002	2.36	2.36	.6920821
105	2.31	2.31	.6774193
109.998	2.27	2.27	.6656892
115.002	2.23	2.23	.653959
120	2.18	2.18	.6392962
150	1.94	1.94	.568915
180	1.74	1.74	.5102639
210	1.56	1.56	.457478
240	1.4	1.40	.4105572
270	1.27	1.27	.372434
300	1.15	1.15	.3372434
330	1.05	1.05	.3079179
360	.96	0.96	.2815249
390	.87	0.87	.255132
420	.8	0.80	.2346041
450	.74	0.74	.2170088
480	.69	0.69	.202346
510	.65	0.65	.1906158
540	.61	0.61	.1788856

570	.58	0.58	.170088
600	.55	0.55	.1612903

CONFINED AQUIFER, FULLY PENETRATING CONDITION

K = 0.2E-03 cm/sec
= 4.5 gpd/ft2
= 0.7E-05 ft/sec
= 0.6 ft/day

REGRESSION COEFFICIENT = -.9959606



X AXIS:
TIME, min

Y AXIS:
LOG (H/H0)

EXAMINE PLOT.
STRIKE ANY KEY WHEN READY.

SRW ASSOCIATES INC.

PROJECT Summit National Site PROJECT NO. 1716

Vertical flow from water table aquifer PAGE 1 OF 1

MADE BY [illegible] DATE 2/20/97 CHECKED BY _____ DATE _____

Heads at wells with wells in shallowest portion of the intermediate units

Well	head ft/R	K (est)	Z
MW-9	1087.11 ft	2×10^{-5} cm/sec	10 ft
MW-10	1086.93		
MW-15	1084.80	6×10^{-7} cm/sec	20 ft
MW-16	1066.35		

Z = vertical distance between midpoints of monitored zones

Area of ~~water table~~ contamination in water-table aquifer

$A \approx 700 \text{ ft} \times 350 \text{ ft} = 245{,}000 \text{ ft}^2$

$Q = KIA$

for wells MW-9 & MW-10:

$$Q = \left(2 \times 10^{-5} \frac{\text{cm}}{\text{sec}} \times \frac{1 \text{ gpd/ft}^2}{4.7 \times 10^{-5} \text{ cm/sec}}\right)\left(\frac{1087.11 \text{ ft} - 1086.93 \text{ ft}}{10 \text{ ft}}\right)(245{,}000 \text{ ft}^2)$$

$$= 4 \times 10^{-1} \text{ gpd/ft}^2 \times 0.018 \times 245000 \text{ ft}^2$$

$$= 1{,}764 \text{ gpd}$$

for wells MW-15 & MW-16

$$Q = \left(6 \times 10^{-7} \frac{\text{cm}}{\text{sec}} \times \frac{1 \text{ gpd/ft}^2}{4.7 \times 10^{-5} \text{ cm/sec}}\right)\left(\frac{1084.80 \text{ ft} - 1066.35 \text{ ft}}{20 \text{ ft}}\right)(245000 \text{ ft}^2)$$

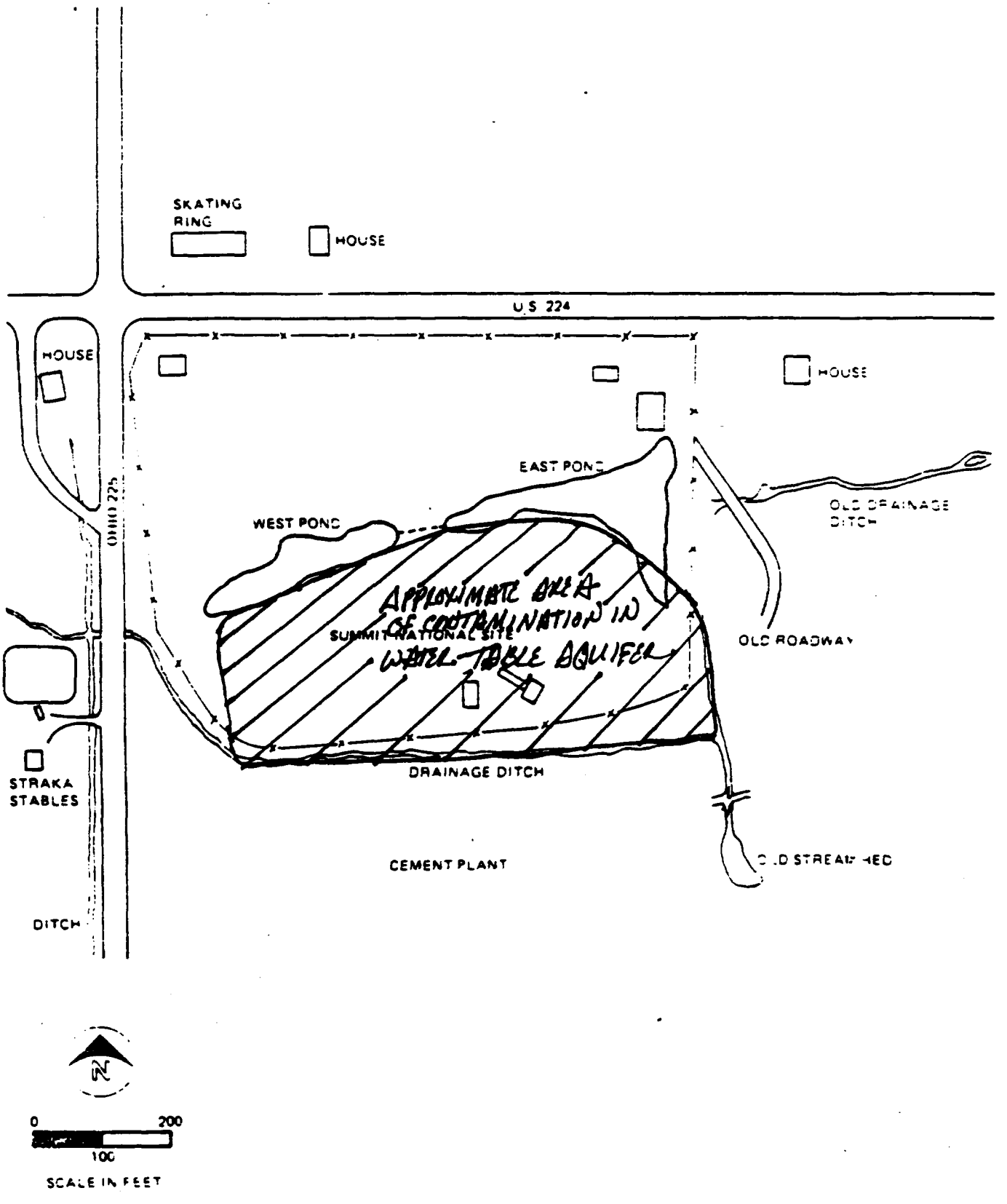
$$= (1 \times 10^{-2} \text{ gpd/ft}^2)(0.685)(245000 \text{ ft}^2)$$

$$= 1{,}612 \text{ gpd}$$

$V = \frac{KI}{n}$ assuming 10% porosity

$$V_{min} = \frac{(6 \times 10^{-7} \text{ cm/sec} / 3.5 \times 10^{-4} \text{ ft/day}) \times 0.7}{0.1} = 0.01 \text{ ft/day}$$

$$V_{max} = \frac{(2 \times 10^{-5} \text{ cm/sec} / 3.5 \times 10^{-4} \text{ ft/day}) \times 0.82}{.1} = 0.01 \text{ ft/day}$$



NOTE: Approximate locations of structures and physical features are approximate

SOURCE: [illegible] - USEPA

SRW ASSOCIATES INC.

PROJECT Summit National Site PROJECT NO. 1316

PAGE 1 OF 1

Calculation of downward flow in intermediate units

MADE BY [illegible] DATE 2/28/89 CHECKED BY _____ DATE _____

$Q = KIA$ $\quad v = \frac{KI}{n}$

$K_{min} = 3 \times 10^{-6}$ cm/sec
$K_{max} = 1 \times 10^{-5}$ cm/sec
} min & max for wells in intermediate units below lowered coal #1a

$A = 245000\ ft^2$ ⇒ area of contamination from calc for downward flow into water-table aquifer

	MW-25	MW-24	Δ
land surface	1076.07 ft	1064.68	11.39
depth (mid-point of monitored zone)	51 ft	64	13

$I = \frac{\Delta h}{\Delta l} = \frac{11.39\ ft}{15\ ft} = 0.9$

$Q_{min} = (3 \times 10^{-6}\ cm/sec / 4.7 \times 10^{-3}\ gpd/ft^2) \times 0.9 \times 245000\ ft^2 = 14,074\ gpd$

$Q_{max} = (1 \times 10^{-5}\ cm/sec / 4.7 \times 10^{-3}\ gpd/ft^2) \times 0.9 \times 245000\ ft^2 = 46,915\ gpd$

assuming $n = 10\%$

$v_{min} = \frac{(3 \times 10^{-6} / 3.5 \times 10^{-4}) \times 0.9}{0.1} = 0.08\ ft/day$

$v_{max} = \frac{(1 \times 10^{-5} / 3.5 \times 10^{-4}) \times 0.9}{0.1} = 0.3\ ft/day$

Weighting for $\frac{K_h}{K_v} = 1.5$ (Freeze & Cherry 1979) $\quad Q = 9400 - 31000\ gpd$

$v = 0.05 - 0.2\ ft/day$

SRW ASSOCIATES INC.

PROJECT Summit National PROJECT NO. 7651

National Fire in Upper Intermediate Units PAGE 1 OF 2

MADE BY [illegible] DATE 2/20/87 CHECKED BY _____ DATE _____

$Q = TIA = T_w I$

I between 1075 & 1088-ft contour lines

$$I = \frac{5\text{ ft}}{(1325 + 1088 - 5)/3}$$

(width between contours @ north, middle, south of contour interval area.

$\bar{w} = 380$ ft

$T = Kb$

b along section between MW-13 and MW-26 = 25 ft, to top of Lower Intermediate Unit

About 1 ft coal, average 4 ft SS. Remainder is silt/st & sh.

$T = T_{coal} + T_{SS} + T_{Slt/st}$

$K_{coal} = 6\times10^{-7} - 6\times10^{-5}$ cm/sec (MW-10 & MW-24)

$K_{SS} = .3\times10^{-6} - 6\times10^{-6}$ cm/sec (MW-22 & MW-12)

$K_{Slt/st} = 9\times10^{-6} - 1\times10^{-5}$ cm/sec (MW-20 & MW-23)

} Use all K_s measured for Intermediate units

$T_{coal} = 1\text{ ft} \times 6\times10^{-7}\text{ cm/sec} \times \frac{1\text{ gpd/ft}^2}{4.7\times10^{-5}\text{ cm/sec}} = 1\times10^{-2}$ gpd/ft min

$= 1 \times 6\times10^{-5} \times \frac{1}{4.7\times10^{-5}} = 1$ gpd/ft max

$T_{SS} = 4 \times 3\times10^{-6} \times \frac{1}{4.7\times10^{-5}} = 3\times10^{-1}$ gpd/ft min

$4 \times 6\times10^{-6} \times \frac{1}{4.7\times10^{-5}} = 5\times10^{-1}$ gpd/ft max

$T_{Slt/st} = 20 \times 9\times10^{-6} \times \frac{1}{4.7\times10^{-5}} = 4$ gpd/ft min

$20 \times 1\times10^{-5} \times \frac{1}{4.7\times10^{-5}} = 4$ gpd/ft max

$T_{max} = 1 + 5\times10^{-1} + 4 = 5$ gpd/ft

$T_{min} = 1\times10^{-2} + 3\times10^{-1} + 4 = 4$ gpd/ft

$Q_{min} = 4$ gpd/ft $\times$ 380 ft $\times 0.05 = 76$ gpd

$Q_{max} = 5$ gpd/ft $\times$ 380 ft $\times 0.05 = 95$ gpd

SRW ASSOCIATES INC.

PROJECT Lateral Flow with Intermediate Limits PROJECT NO. 7215

PAGE 2 OF 2

MADE BY [illegible] DATE 2/20/89 CHECKED BY _____ DATE _____

$$V = \frac{KI}{n}$$

assuming $n = 10\%$

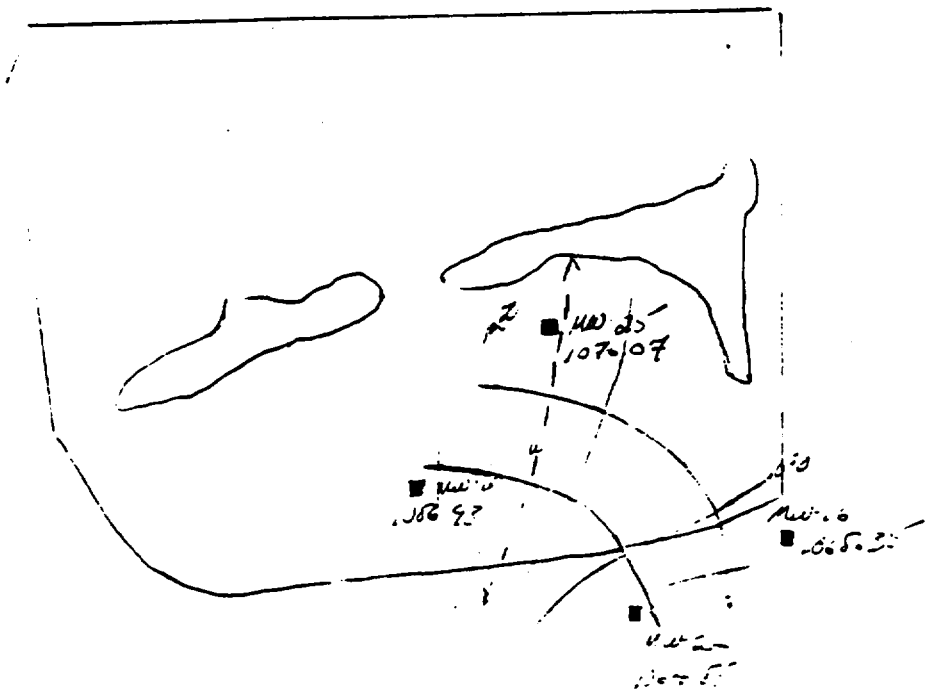
& $K = 6 \times 10^{-7}$ cm/sec (in coal, MW-16) to 6×10^{-5} cm/sec (in sa., MW-15)

$$V_{min} = \frac{(6 \times 10^{-7}\ \text{cm/sec} \cdot \frac{1\ \text{ft/day}}{3.5 \times 10^{-4}\ \text{cm/sec}}) \times 0.05}{10\%}$$

$$= 9 \times 10^{-4}\ \text{ft/day} = 0.3\ \text{ft/yr}$$

$$V_{max} = \frac{(6 \times 10^{-5}\ \text{cm/sec} \times \frac{1\ \text{ft/day}}{3.5 \times 10^{-4}\ \text{cm/sec}}) \times 0.05}{10\%} =$$

$$= 9 \times 10^{-2}\ \text{ft/day} = 31\ \text{ft/yr}$$



Flow in upper intermediate units

SRW ASSOCIATES INC.

PROJECT Summit Natural Site — PROJECT NO. 8535

Fateful Plain Lower Intermediate Units — PAGE 1 OF 2

MADE BY [illegible] DATE 2/20/87 CHECKED BY _____ DATE _____

$Q = KIA = TwI$

I between 1064 and 1065-ft contour lines

$I = \frac{1\,ft}{75\,ft} = 1 \times 10^{-2}$

$w = 340\,ft$

$T = Kb$

Balmy sands in MW-15 & MW-26 ≈ 32ft. Average ~2ft cal, ~3ft ss.
Remainder = SHst/sh

$T = T_{cal} + T_{ss} + T_{SHst}$

$K_{cal} = 6 \times 10^{-7} - 6 \times 10^{-5}$ cm/sec (MW-10 + MW-24) } using all
$K_{ss} = 3 \times 10^{-6} - 6 \times 10^{-4}$ cm/sec (MW-22 + MW-12) } K_s measured
$K_{SHst} = 9 \times 10^{-6} - 1 \times 10^{-5}$ cm/sec (MW-20 + MW-23) } for intermediate units

$T_{cal} = 2\,ft \times 6 \times 10^{-7}$ cm/sec $\times \frac{1\,gpd/ft^2}{4.7 \times 10^{-5}\,cm/sec} = 3 \times 10^{-2}$ gpd/ft min

$2\,ft \times 6 \times 10^{-5}$ cm/sec $\times \frac{1\,gpd/ft^2}{4.4 \times 10^{-7}\,cm/sec} = 3$ gpd/ft max

$T_{ss} = 3 \times 3 \times 10^{-6} \times \frac{1}{4.7 \times 10^{-5}} = 2 \times 10^{-1}$ gpd/ft min

$3 \times 6 \times 10^{-4} \times \frac{1}{4.7 \times 10^{-5}} = 4 \times 10^{-1}$ gpd/ft max

$T_{SHst} = 27 \times 9 \times 10^{-6} \times \frac{1}{4.7 \times 10^{-5}} = 5$ gpd/ft min

$27 \times \frac{1 \times 10^{-5}}{9 \times 10^{-6}} \times \frac{1}{4.7 \times 10^{-5}} = 6$ gpd/ft max

$T_{min} = 3 \times 10^{-2} + 2 \times 10^{-1} + 5 = 5$ gpd/ft

$T_{max} = 3 + 4 \times 10^{-1} + 6 = 9$ gpd/ft

$Q_{min} = 5\,gpd/ft \times 340\,ft \times 0.01 = 17$ gpd

$Q_{max} = 9\,gpd/ft \times 340\,ft \times 0.01 = 31$ gpd

SRW ASSOCIATES INC.

PROJECT Summit PROJECT NO. 836

Lateral Flow in Lower Intermediate Units PAGE 2 OF 2

MADE BY [illegible] DATE 2/20/87 CHECKED BY _____ DATE _____

$$V = \frac{KI}{n}$$

assuming $n = 10\%$

$K_{min} = 6 \times 10^{-7}$ cm/sec

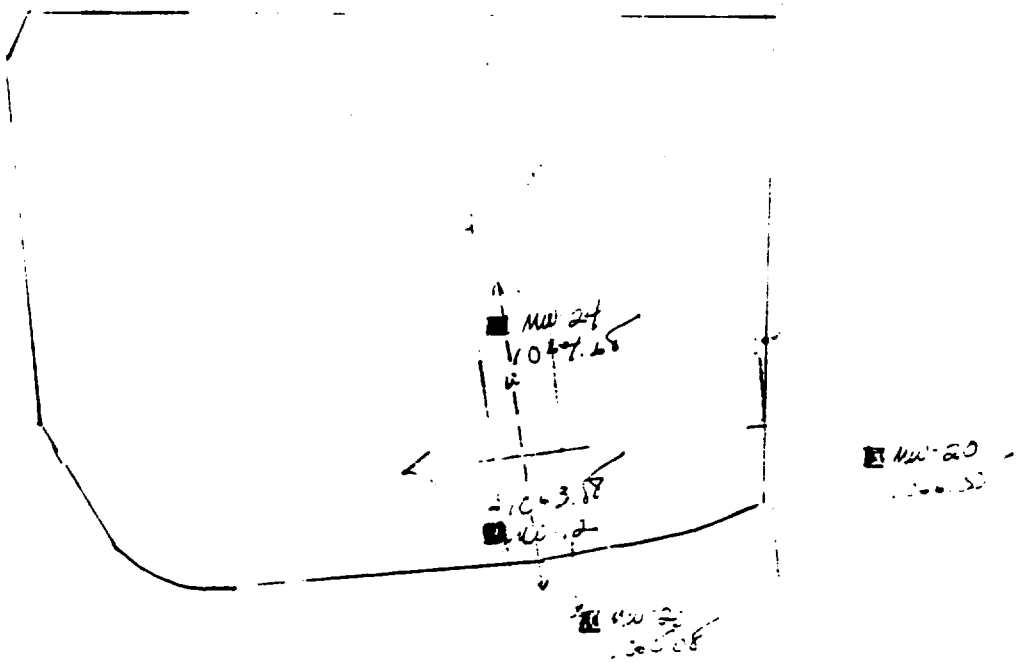
$K_{max} = 6 \times 10^{-5}$ cm/sec

$$V_{min} = \frac{(6\times10^{-7}\ \text{cm/sec} \times \frac{1\ \text{ft/day}}{3.5\times10^{-4}\ \text{cm/sec}}) \times 0.01}{10\%}$$

$= 2 \times 10^{-4}$ ft/day $= 6 \times 10^{-2}$ ft/yr

$$V_{max} = \frac{(6\times10^{-5}\ \text{cm/sec} \times \frac{1\ \text{ft/day}}{3.5\times10^{-4}\ \text{cm/sec}}) \times 0.01}{10\%}$$

$= 2 \times 10^{-2}$ ft/day $= 6$ ft/yr



Flow in Area Immediate
Limits

E

SRW ASSOCIATES INC.

PROJECT Summit National Site PROJECT NO. 8635

PAGE 1 OF 1

Calculation of Flow in Upper Zone

MADE BY [illegible] DATE 12/24/87 CHECKED BY _____ DATE _____

$Q = KIA$

$I = \frac{2\,ft}{(250 + 210 + 285)/3} = \frac{2}{252} = 8 \times 10^{-3}$ (see attached)

↑ average flow length

$A = w \times b$

$b = 45\,ft$

$w = 710'$

$A = 45\,ft \times 710\,ft = 31{,}950\,ft^2$

$K_{min} = 5 \times 10^{-5}\,cm/sec = 1\,gpd/ft^2$ (MW-14)

$K_{max} = 3 \times 10^{-4}\,cm/sec = 6\,gpd/ft^2$ (MW-5)

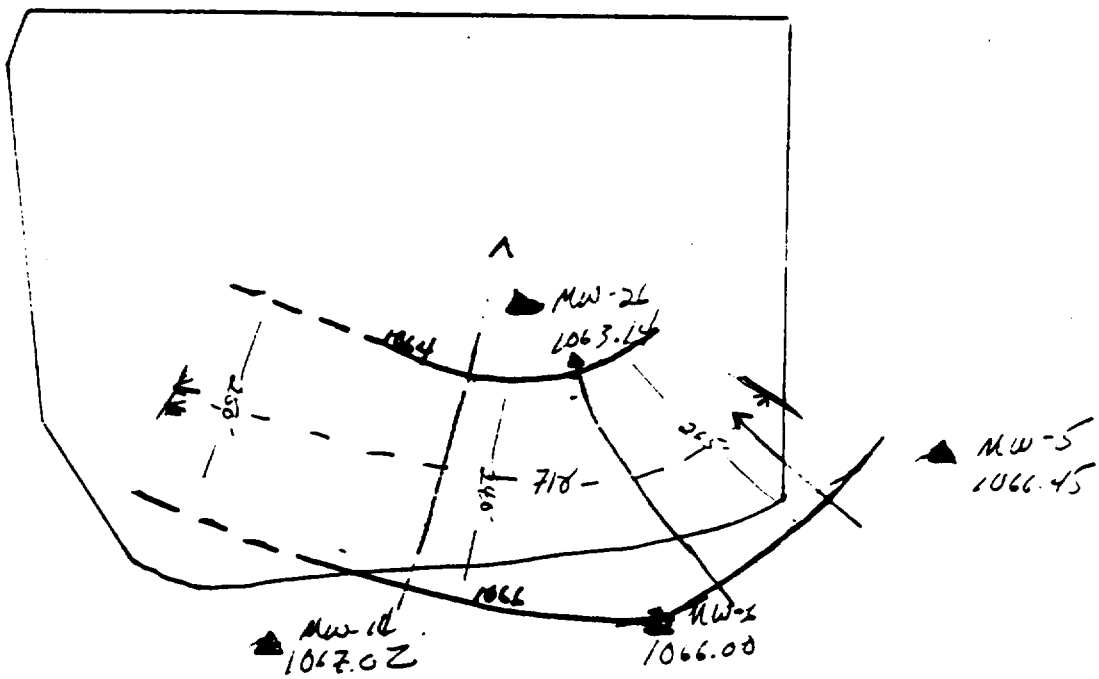
$Q_{min} = 1\,gpd/ft^2 \times (8 \times 10^{-3}) \times 31{,}950\,ft^2 = \frac{251}{\cancel{200}}\,gpd$

$Q_{max} = 6\,gpd/ft^2 \times (8 \times 10^{-3}) \times 31{,}950\,ft^2 = 1534\,gpd$

$V = \frac{KI}{n}$ assuming $n = 10\%$

$V_{min} = \frac{1\,gpd/ft\,(8 \times 10^{-3})}{7.48\,\frac{gal}{ft^3}\;10\%} = \frac{1 \times 10^{-2}}{\cancel{1 \times 10^{-2}}}\,ft/dy$

$V_{max} = \frac{6\,gpd/ft\,(8 \times 10^{-3})}{7.48\,\frac{gal}{ft^3}\;10\%} = \frac{0.06}{\cancel{0.6}}\,ft/dy$



LEGEND:

▲ MONITORING WELL

1069.21 HYDRAULIC HEAD (FT.)

EQUIPOTENTIAL LINE, DASHED WHERE TENTATIVE
(CONTOUR INTERVAL = 2 FT.)

FLOW LINE

~~Monitoring well with discharge~~

NOTE: All features of structures and physical features approximate.

SOURCE: Modified from USEPA.

SCALE IN FEET
APPROXIMATE

FIGURE 4-~~2~~6
PIEZOMETRIC SURFACE ON THE UPPER SHARON AQUIFER
SUMMIT NATIONAL RI

CALCULATION OF GROUNDWATER FLOW QUANTITIES IN WATER-TABLE AQUIFER
SUMMIT NATIONAL SITE DBT
27-Feb-97

Q = KIA = K (dh/dl) x w x b
v = KI/n

Q = total flow q = flow in single cell
dh = change in head dl = flow length
w = width of flow cell b = aquifer thickness
I = dh/dl n = porosity
K = hydraulic conductivity (assumed from typical values for these materials)

CALCUALTION FOR HIGH-END HYDRAULIC CONDUCTIVITY
Using K(fill) = 3E-3 cm/sec. (MW-3 - maximum valid slug test for fill)
K(till) = 2E-3 cm/sec (MW-7 - maximum valid slug test for till)
K(rock) = 1E-5 from MW-2, closest bedrock well to area used for calculation.
Performing calculations between 1084 and 1086 water-table contour lines

Calculated val:

CELL	w (ft)	dl (ft)	b (ft)	I = 2/dl	K (cm/sec)	K (gpd/ft2)	K (ft/d)	q (gpd)	n	v (ft/d)
A	150	25	23	0.08	3E-03	64	9	17542	40%	1.71
B	135	30	24	0.066666	3E-03	64	9	13729	40%	1.43
C	70	50	21	0.04	3E-03	64	9	3737	40%	0.86
D	45	90	22	0.022222	3E-03	64	9	1398	40%	0.48
E	80	65	22	0.030769	3E-03	64	9	3442	40%	0.66
F	80	50	22	0.04	3E-03	64	9	4475	40%	0.86
G	120	70	14	0.028571	3E-03	64	9	3051	40%	0.61
H	75	60	10	0.033333	3E-03	64	9	1589	40%	0.71
I	85	20	10	0.1	3E-03	64	9	5403	40%	2.14
J	125	25	15	0.08	3E-03	64	9	9534	40%	1.71
K(till)	100	35	8	0.057142	2E-03	42	6	1937	30%	1.09
K(rock)	100	35	12	0.057142	1E-05	0.2	0.03	15	20%	0.01
L(till)	110	50	13	0.04	2E-03	42	6	2424	30%	0.76
L(rock)	110	50	12	0.04	1E-05	0.2	0.03	11	20%	0.01
								=========		=========
						TOTAL FLOW (gpd)		68,286		
						(gpm)		47		
							AVERAGE FLOW RATE			0.93

CALCULATION FOR LOW-END HYDRAULIC CONDUCTIVITY

Using K(fill) = 1E-4 cm/sec. (MW-15 - minimum valid slug test for fill)
K(till) = 6E-5 cm/sec (MW-17 - minimum valid slug test for till)
K(rock) = 1E-5 from MW-2, closest bedrock well to area used for calculation.
Performing calculations between 1084 and 1086 water-table contour lines

Calculated val:

CELL	w (ft)	dl (ft)	b (ft)	* I = 2/dl	K (cm/sec)	K (gpd/ft2)	K (ft/d)	* q (gpd)	n	* v (ft/s)
A	150	25	23	0.08	1E-04	2	0.3	585	40%	0.06
B	135	30	24	0.066666	1E-04	2	0.3	458	40%	0.05
C	70	50	21	0.04	1E-04	2	0.3	125	40%	0.02
D	45	90	22	0.022222	1E-04	2	0.3	47	40%	0.01
E	80	65	22	0.030769	1E-04	2	0.3	115	40%	0.02
F	80	50	22	0.04	1E-04	2	0.3	149	40%	0.03
G	120	70	14	0.028571	1E-04	2	0.3	102	40%	0.02
H	75	60	10	0.033333	1E-04	2	0.3	53	40%	0.02
I	85	20	10	0.1	1E-04	2	0.3	180	40%	0.07
J	125	25	15	0.08	1E-04	2	0.3	318	40%	0.06
K(till)	100	35	8	0.057142	6E-05	1	0.2	58	30%	0.02
K(rock)	100	35	12	0.057142	1E-05	0.2	0.03	15	20%	0.00
L(till)	110	50	13	0.04	6E-05	1	0.2	73	30%	0.02
L(rock)	110	50	12	0.04	1E-05	0.2	0.03	11	20%	0.00
								=========		=========
						TOTAL FLOW	(gpd)	2,287		
							(gpm)	2		
								AVERAGE FLOW RATE		0.03

Summit

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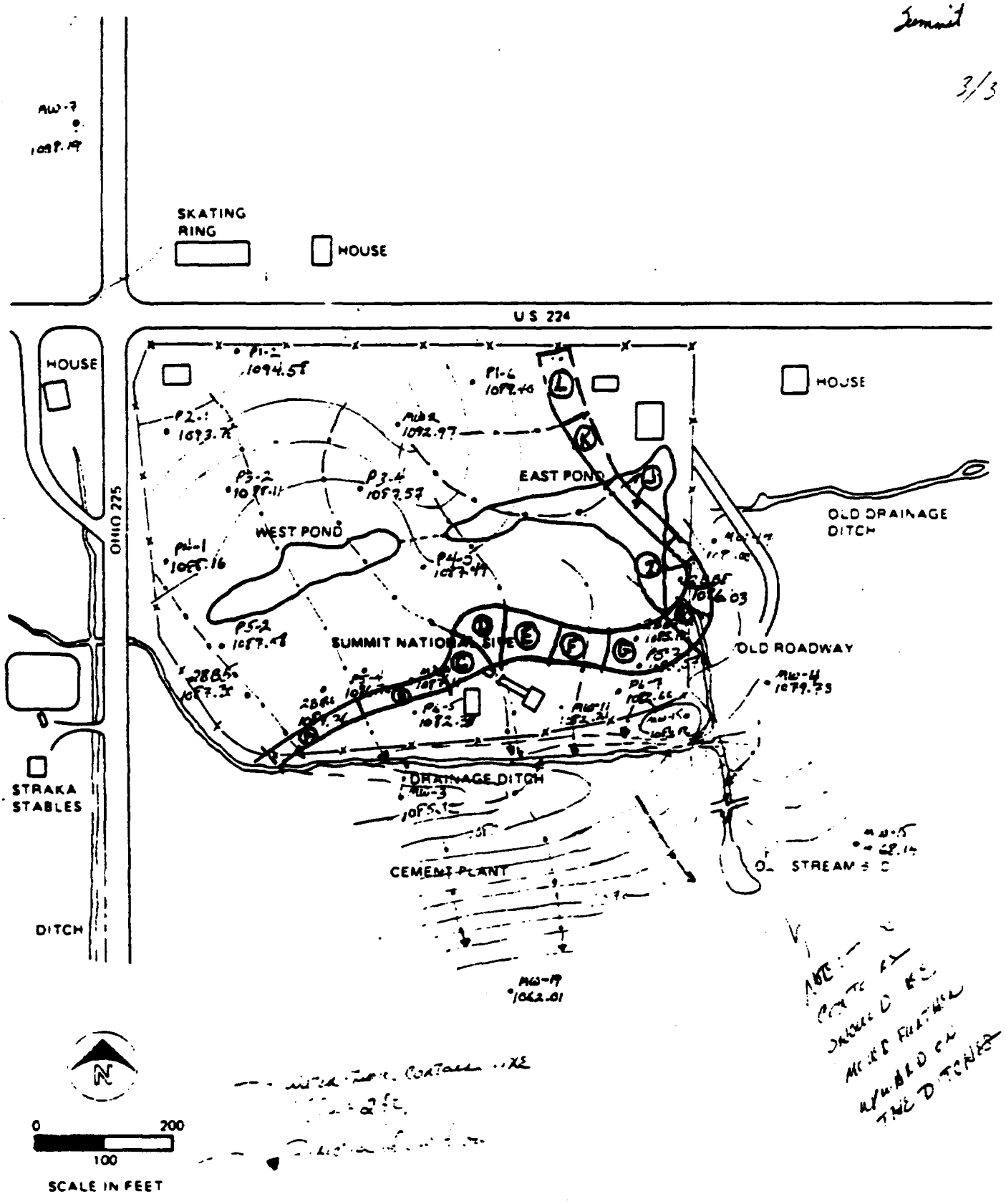

Water-Table Map
Sept., 1986

NOTE All locations of structures and physical features approximate

SOURCE Modified from USEPA

Project: Summit National Site
Flow and transport calculations for water-table aquifer
By: DBT Date: 26-Feb-87
Checked: _____ Date: _____
Filename: WTI80862.WK1

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Retardation coefficients and adjusted times for previous concentrations:
Assuming 40% porosity, 1% organic carbon, density of 100 lb/ft3 (2 g/cm3)
References: Practical Aspects of Groundwater Modeling, W.C. Walton, 1984.
Handbook of Env. Data on Organic Chemicals, 2nd ed, K. Verschueren, 1983.
(Kow not available for all compounds)
Kd = 6.3E-1 x f x Kow (distribution coefficient ml/g)
f = organic carbon content
Kow = octanol-water partitioning coefficient
Rd = 1 +(ps/n x Kd) (retardation factor, dimensionless)
ps = dry bulk density
n = porosity

COMPOUND	Kow (molar)	Kd (ml/g)	Rd
Methylene chloride	8.12	0.05	1.3
1,1-Dichloroethane	61.7	0.39	2.9
1,2-Dichloroethane	30.2	0.19	2.0
2-Butanone	-0.059	-0.00	1.0
1,1,1-Trichloroethane	148	0.93	5.7
Trichloroethene	195	1.23	7.1
Toluene	490	3.09	16.4
Ethylbenzene	3.15	0.02	1.1
Phenol	28.8	0.18	1.9
Isophorone	0.23	0.001	1.0
2,4-Diethylphenol	2.5	0.02	1.1
Naphthalene	34.4	0.22	2.1
Bis(2-ethylhexyl)phthalate	540000000	3.40E+06	1.70E+07

HIGH-VELOCITY CASE

a = 20 ft
n = 0.4
V = 0.9 ft/day
ax = 60 ft (Walton, 1984)
ay = 10 ft (Walton, 1984)
az = 10 ft (Walton, 1984)
Do assumed as 0

Dx = 54 ft2/d
Dy = 9 ft2/d
Dz = 9 ft2/d

References: Domenico and Robbins, 1985
Gilham, 1982

Source dimensions: X = 110 ft
Y = 440 ft
Z = 20 ft

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D

(edit)(date)(dcom)

for location x=100, y=0, z=0
time = 10 years

CONTAMINANT	Bd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene chloride	1.25578	0.71668604	43.0011626	7.17E+00	7.16688604	-3.1756472		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	24,000	4,080
1,1-Dichloroethane	2.94355	0.30575325	18.3451954	3.06E+00	3.05753325	-1.9631606		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	12,000	2,040
1,2-Dichloroethane	1.9513	0.46123097	27.6738584	4.61E+00	4.6123097	-2.4911786		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	115,000	19,550
2-Butanone	0.9581415	0.90167576	54.1005458	9.02E+00	9.0167576	-3.5906866		1.93	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	650,000	110,500
1,1,1-Trichloroethane	5.662	0.15895443	9.53726598	1.59E+00	1.5895443	-1.2668256		1.88	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	53,000	9,010
Trichloroethene	7.1425	0.12600630	7.56037801	1.26E+00	1.2600630	-1.0833316		1.48	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	18,000	3,060
Toluene	16.435	0.05476118	3.28567082	5.48E-01	0.5476118	-0.4360187		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	27,000	4,562
Ethylbenzene	1.099225	0.81875867	49.1255202	8.19E+00	8.1875867	-3.4106544		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	11,000	1,870
Phenol	1.9072	0.47189597	28.3137583	4.72E+00	4.7189597	-2.5234121		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	7,000	1,190
Isophorone	1.007245	0.89352640	53.6115840	8.94E+00	8.9352640	-3.5733063		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	26,000	4,420
2,4-Dimethylphenol	1.07875	0.83429895	50.0579374	8.34E+00	8.3429895	-3.4450901		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	140	24
Naphthalene	2.0836	0.43194471	25.9166826	4.32E+00	4.3194471	-2.4004635		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	620	105
Bis(2-ethylhexyl)phthalate	17010001	5.29E-08	3.17E-06	5.29E-07	5.29E-07	464.491769		0	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	1,250	0

for location x=100, y=0, z=0
time = 50 years

CONTAMINANT	Bd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene chloride	1.25578	0.71668604	43.0011626	7.17E+00	7.16688604	-7.3258346		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	24,000	4,080
1,1-Dichloroethane	2.94355	0.30575325	18.3451954	3.06E+00	3.0575325	-4.7534113		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	12,000	2,040
1,2-Dichloroethane	1.9513	0.46123097	27.6738584	4.61E+00	4.6123097	-5.6518705		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	115,000	19,550
2-Butanone	0.9581415	0.90167576	54.1005458	9.02E+00	9.0167576	-8.2300745		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	650,000	110,500
1,1,1-Trichloroethane	5.662	0.15895443	9.53726598	1.59E+00	1.5895443	-3.3568124		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	53,000	9,010
Trichloroethene	7.1425	0.12600630	7.56037801	1.26E+00	1.2600630	-2.9608300		1.99	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	18,000	3,060
Toluene	16.435	0.05476118	3.28567082	5.48E-01	0.5476118	-1.8364140		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	27,000	4,562
Ethylbenzene	1.099225	0.81875867	49.1255202	8.19E+00	8.1875867	-7.8376800		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	11,000	1,870
Phenol	1.9072	0.47189597	28.3137583	4.72E+00	4.7189597	-5.9207483		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	7,000	1,190
Isophorone	1.007245	0.89352640	53.6115840	8.94E+00	8.9352640	-8.1963414		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	26,000	4,420
2,4-Dimethylphenol	1.07875	0.83429895	50.0579374	8.34E+00	8.3429895	-7.9127041		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	140	24
Naphthalene	2.0836	0.43194471	25.9166826	4.32E+00	4.3194471	-5.6584227		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	620	105
Bis(2-ethylhexyl)phthalate	17010001	5.29E-08	3.17E-06	5.29E-07	5.29E-07	207.725429		0	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	1,250	0

for location x=100, y=0, z=0
time = 100 years

CONTAMINANT	Bd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene chloride	1.25578	0.71668604	43.0011626	7.17E+00	7.16688604	-10.400204		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	24,000	4,080
1,1-Dichloroethane	2.94355	0.30575325	18.3451954	3.06E+00	3.0575325	-6.7579859		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	12,000	2,040
1,2-Dichloroethane	1.9513	0.46123097	27.6738584	4.61E+00	4.6123097	-8.3633441		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	115,000	19,550
2-Butanone	0.9581415	0.90167576	54.1005458	9.02E+00	9.0167576	-11.674664		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	650,000	110,500
1,1,1-Trichloroethane	5.662	0.15895443	9.53726598	1.59E+00	1.5895443	-4.8319942		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	53,000	9,010
Trichloroethene	7.1425	0.12600630	7.56037801	1.26E+00	1.2600630	-4.2824273		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	0.17	18,000	3,060
Toluene	16.435	0.05476118	3.28567082	5.48E-01	0.5476118	-2.7414914		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	27,000	4,590
Ethylbenzene	1.099225	0.81875867	49.1255202	8.19E+00	8.1875867	-11.121493		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	0.17	11,000	1,870
Phenol	1.9072	0.47189597	28.3137583	4.72E+00	4.7189597	-8.4223867		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	-0.17	7,000	1,190
Isophorone	1.007245	0.89352640	53.6115840	8.94E+00	8.9352640	-11.621463		2	3.47850542	1	-3.4785054	-1	0.15811388	0.17	-0.1581138	0.17	26,000	4,420

2,4-Dimethylphenol	1.07875	0.834298955	50.05793174	8.34E+00	8.34298955	11.22724	2	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.15811388	-0.17	140	24
Naphthalene	2.0836	0.43194471	25.91668426	4.32E+00	4.31944471	-8.05364265	2	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.15811388	-0.17	620	105
Bis(2-ethylhexyl)phthalate	17010001	5.29E-08	3.17E-06	5.29E-07	5.29E-07	146.8826841	0	3.47850542	1	-3.47850542	-1	0.15811388	0.17	0.15811388	0.17	7,250	0

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Retardation coefficients and adjusted times for previous concentrations:
Assuming 40% porosity, 1% organic carbon, density of 100 lb/ft3 (1 g/cm3)
References: Practical Aspects of Groundwater Modeling, D.C. Walton, 1984.
Handbook of Env. Data on Organic Chemicals, 2nd ed, K. Verschueren, 1983.
(Koc not available for all compounds)
Kd = 6.3E-1 x f x Koc (distribution coefficient ml/g)
f = organic carbon content
Koc = octanol-water partitioning coefficient
Rd = 1 +(ps/n x Kd) (retardation factor, dimensionless)
ps = dry bulk density
n = porosity

COMPOUND	Koc (molar)	Kd (ml/g)	Rd
Methylene chloride	8.12	0.05	1.3
1,1-Dichloroethane	61.7	0.39	2.9
1,2-Dichloroethane	30.2	0.19	2.0
2-Butanone	-0.659	-0.00	1.0
1,1,1-Trichloroethane	148	0.93	5.7
Trichloroethene	195	1.23	7.1
Toluene	490	3.09	16.4
Ethylbenzene	3.15	0.02	1.1
Phenol	28.8	0.18	1.9
Isophorone	0.23	0.001	1.0
2,4-Dimethylphenol	2.5	0.02	1.1
Naphthalene	34.4	0.22	2.1
Bis(2-ethylhexyl)phthalate	540000000	3.40E+06	1.70E+07

[illegible] VELOCITY CASE

n = 20 ft
n = 0.4
V = 0.03 ft/day
ax = 60 ft (Walton, 1984)
ay = 10 ft (Walton, 1984)
az = 10 ft (Walton, 1984)
Do assumed as 0

Dx = 1.8 ft2/d
Dy = 0.3 ft2/d
Dz = 0.3 ft2/d

References: Domenico and Robbins, 1985
Gilham, 1982

Source dimensions: X = 110 ft
Y = 440 ft
Z = 20 ft

for location x=100, y=0, z=0 (edit)(absl)(down)
time = 10 years

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene chloride	1.2557	0.0238853	1.4333/208	2.15E-01	0.2388953	0.0885393	0.89	3.4785054	1	-3.478504	1	0.1581388	0.17	0.1581138	-0.17	29,000	1,816

1,1-Dichloroethane	2.94355	0.01019177	0.61150651	1.0[illegible]-01	0.1019177	0.66463439	0.36	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	0.17	1[illegible],000	567
1,2-Dichloroethane	1.9513	0.01537436	0.92246194	1.5[illegible]-01	0.1537436	0.37813879	0.57	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	115,000	3,572
2-Butanone	0.9381415	0.03005585	1.80335152	3.01E-01	0.3005585	-0.059803B	1.06	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	630,000	58,565
1,1,1-Trichloroethane	5.662	0.00529848	0.31790886	5.30E-02	0.0529848	1.18455010	0.09	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	53,000	405
Trichloroethene	7.1425	0.00420021	0.25201260	4.20E-02	0.0420021	1.39584781	0.05	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	18,000	77
Toluene	16.433	0.00185537	0.10952246	1.8[illegible]-02	0.0185537	2.33414456	0.001	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	27,000	5
Ethylbenzene	1.095225	0.02729195	1.63751734	2.7[illegible]-01	0.2729195	0.00248582	1	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	11,000	935
Phenol	1.9072	0.01572986	0.94379194	1.5[illegible]-01	0.1572986	0.36278739	0.62	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	7,000	569
Isophorone	1.007245	0.02978421	1.78705280	2.98E-01	0.2978421	-0.0539376	1.06	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	26,000	2,343
2,4-Dimethylphenol	1.07875	0.02780996	1.66859791	2.78E-01	0.2780996	-0.0096511	1	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	140	12
Naphthalene	2.0836	0.01439815	0.86388942	1.44E-01	0.1439815	0.46284474	0.57	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	620	50
Bis(2-ethylhexyl)phthalate	17010001	1.76E-09	1.06E-07	1.76E-08	1.76E-08	2544.13094	0	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	7,250	0

for location x=100, y=0, z=0
time = 50 years

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene chloride	1.25578	0.02388553	1.43337208	2.39E-01	0.2388553	-1.0386702	1.84	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	24,000	3,754
1,1-Dichloroethane	2.94355	0.01019177	0.61150651	1.0[illegible]-01	0.1019177	-0.4070389	1.43	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	1[illegible],000	1,459
1,2-Dichloroethane	1.9513	0.01537436	0.92246194	1.5[illegible]-01	0.1537436	-0.6958877	1.68	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	115,000	16,922
2-Butanone	0.9381415	0.03005585	1.80335152	3.01E-01	0.3005585	-1.2361740	1.91	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	630,000	105,548
1,1,1-Trichloroethane	5.662	0.00529848	0.31790886	5.30E-02	0.0529848	0.02167998	1	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	53,000	4,505
Trichloroethene	7.1425	0.00420021	0.25201260	4.20E-02	0.0420021	0.17212463	0.83	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	18,000	1,270
Toluene	16.433	0.00185537	0.10952246	1.8[illegible]-02	0.0185537	0.74580968	0.29	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	27,000	666
Ethylbenzene	1.095225	0.02729195	1.63751734	2.73E-01	0.2729195	-1.1513675	1.91	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	11,000	1,786
Phenol	1.9072	0.01572986	0.94379194	1.57E-01	0.1572986	-0.7126966	1.68	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	7,000	1,000
Isophorone	1.007245	0.02978421	1.78705280	2.98E-01	0.2978421	-1.2280727	1.91	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	26,000	4,221
2,4 Dimethylphenol	1.07875	0.02780996	1.66859791	2.78E-01	0.2780996	-1.1676816	1.91	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	140	23
Naphthalene	2.0836	0.01439815	0.86388942	1.44E-01	0.1439815	-0.6481479	1.64	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	0.17	620	86
Bis(2-ethylhexyl)phthalate	17010001	1.76E-09	1.06E-07	1.76E-08	1.76E-08	1137.76965	0	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	0.17	7,250	0

for location x=100, y=0, z=0
time = 100 years

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene chloride	1.25578	0.02388553	1.43337208	2.39E-01	0.2388553	-1.6874985	1.98	3.47850542	1	-3.47850542	1	0.15811388	0.17	-0.1581138	0.17	24,000	4,039
1,1-Dichloroethane	2.94355	0.01019177	0.61150651	1.0[illegible]-01	0.1019177	-0.9103148	1.8	3.47850542	1	-3.47850542	1	0.15811388	0.17	-0.1581138	0.17	1[illegible],000	1,836
1,2-Dichloroethane	1.9513	0.01537436	0.92246194	1.5[illegible]-01	0.1537436	1.2564231	1.93	3.47850542	1	-3.47850542	1	0.15811388	0.17	-0.1581138	0.17	115,000	18,866
2-Butanone	0.9381415	0.03005585	1.80335152	3.01E-01	0.3005585	-1.9431013	1.99	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	630,000	109,948
1,1,1-Trichloroethane	5.662	0.00529848	0.31790886	5.30E-02	0.0529848	-0.4335047	1.48	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	0.17	53,000	6,867
Trichloroethene	7.1425	0.00420021	0.25201260	4.20E-02	0.0420021	-0.2779086	1.33	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	0.17	18,000	2,015
Toluene	16.433	0.00185537	0.10952246	1.8[illegible]-02	0.0185537	0.26392412	0.72	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	-0.17	27,000	1,652
Ethylbenzene	1.095225	0.02729195	1.63751734	2.73E-01	0.2729195	-1.8327972	1.99	3.47850542	1	-3.47850542	-1	0.15811388	0.17	-0.1581138	0.17	11,000	1,861
Phenol	1.9072	0.01572986	0.94379194	1.57E-01	0.1572986	-1.2772976	1.93	3.47850542	1	-3.47850542	1	0.15811388	0.17	-0.1581138	0.17	7,000	1,148
Isophorone	1.007245	0.02978421	1.78705280	2.98E-01	0.2978421	-1.9523511	1.99	3.47850542	1	-3.47850542	1	0.15811388	0.17	-0.1581138	0.17	26,000	4,398
2,4 Dimethylphenol	1.07875	0.02780996	1.66859791	2.78E-01	0.2780996	-1.8539544	1.99	3.47850542	1	-3.47850542	1	0.15811388	0.17	-0.1581138	0.17	140	24
Naphthalene	2.0836	0.01439815	0.86388942	1.44E-01	0.1439815	-1.1981948	1.97	3.47850542	1	-3.47850542	1	0.15811388	0.17	-0.1581138	0.17	620	101
Bis(2-ethylhexyl)phthalate	17010001	1.76E-09	1.06E-07	1.76E-08	1.76E-08	804.5249380	0	3.47850542	1	-3.47850542	1	0.15811388	0.17	-0.1581138	0.17	7,250	0

v= 0.9 ft/day
time= 36500 days
x= 1450 feet

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.7166860	43.00116	7.166860	7.166860	-9.86141	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	24,000	1,079
1,1-Dichloroethane	2.94355	0.3057532	18.34519	3.057532	3.057532	-5.93309	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	12,000	539
1,2-Dichloroethane	1.9513	0.4612309	27.67385	4.612309	4.612309	-7.65392	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	115,000	5,169
2-Butanone	0.998141	0.9016757	54.10054	9.016757	9.016757	-11.1943	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	650,000	29,214
1,1,1-Trichloroethane	5.662	0.1589544	9.537265	1.589544	1.589544	-3.68794	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	53,000	2,382
Trichloroethene	7.1425	0.1260063	7.560378	1.260063	1.260063	-2.99747	1.9999	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	18,000	809
Toluene	16.435	0.0547611	3.285670	0.547611	0.547611	-0.79234	1.7421	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	27,000	1,057
Ethyl Benzene	1.099225	0.8187586	49.12552	8.187586	8.187586	-10.6174	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	11,000	494
Phenol	1.9072	0.4718959	28.31375	4.718959	4.718959	-7.75840	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	7,000	315
Isophorone	1.007245	0.8935264	53.61158	8.935264	8.935264	-11.1389	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	26,000	1,169
2,4-Dimethylphenol	1.07875	0.8342989	50.05793	8.342989	8.342989	-10.7278	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	140	6
Naphthalene	2.0816	0.4319447	25.91668	4.319447	4.319447	-7.35961	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	620	28
Bis(2-ethylhexyl)phthalat	17010001	0.0000000	0.000003	0.000000	0.000000	2129.836	0	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	7,250	0

SUMMIT NATIONAL SITE

Project: Summit National Site
Contaminant Transport Calculations for Water Table Aquifer

By: DBT Date: 10 Dec 87

SEE TRANSPORT CALCULATIONS OF 2/26/87 FOR TERMS, EQUATIONS AND REFERENCES

HIGH VELOCITY CASE

v= 0.9 ft/day
time= 3650 days
x= 1450 feet

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.7166860	43.00116	7.166860	7.166860	-1.47145	1.966	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	24,000	1,060
1,1-Dichloroethane	2.94355	0.3057532	18.34519	3.057532	3.057532	0.645371	0.3579	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	12,000	97
1,2-Dichloroethane	1.9513	0.4612309	27.67385	4.612309	4.612309	-0.36733	1.3794	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	115,000	3,565
2-Butanone	0.998141	0.9016757	54.10054	9.016757	9.016757	-2.07159	1.997	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	650,000	29,171
1,1,1-Trichloroethane	5.662	0.1589544	9.537265	1.589544	1.589544	2.330983	0.00114	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	53,000	1
Trichloroethene	7.1425	0.1260063	7.560378	1.260063	1.260063	2.980031	0.000022	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	18,000	0
Toluene	16.435	0.0547611	3.285670	0.547611	0.547611	5.707735	0	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	27,000	0
Ethyl Benzene	1.099225	0.8187586	49.12552	8.187586	8.187586	-1.81659	1.9891	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	11,000	492
Phenol	1.9072	0.4718959	28.31375	4.718959	4.718959	-0.42370	1.4284	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	7,000	225
Isophorone	1.007245	0.8935264	53.61158	8.935264	8.935264	-2.04739	1.9953	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	26,000	1,166
2,4-Dimethylphenol	1.07875	0.8342989	50.05793	8.342989	8.342989	-1.86594	1.9928	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	140	6
Naphthalene	2.0836	0.4319447	25.91668	4.319447	4.319447	-0.20580	1.2227	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	620	17
Bis(2-ethylhexyl)phthalat	17010001	0.0000000	0.000003	0.000000	0.000000	6735.142	0	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	-0.0564	7,250	0

v= 0.9 ft/day
time= 18250 days
x= 1450 feet

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.7166860	43.00116	7.166860	7.166860	-6.56387	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	0.0564	24,000	1,079
1,1 Dichloroethane	2.94355	0.3057532	18.34519	3.057532	3.057532	-3.56884	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	-0.04152	0.0564	12,000	539
1,2-Dichloroethane	1.9513	0.4612309	27.67385	4.612309	4.612309	-4.90205	2	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	0.04152	0.0564	115,000	5,169
2-Butanone	0.998141	0.9016757	54.10054	9.016757	9.016757	-7.55075	2	0.913500	0.7969	0.91350	-0.7969	0.041522	0.0564	0.04152	0.0564	650,000	29,214
1,1,1-Trichloroethane	5.662	0.1589544	9.537265	1.589544	1.589544	-1.73887	1.9838	0.913500	0.7969	-0.91350	-0.7969	0.041522	0.0564	0.04152	0.0564	53,000	2,363
Trichloroethene	7.1425	0.1260063	7.560378	1.260063	1.260063	-1.14363	1.8802	0.913500	0.7969	0.91350	-0.7969	0.041522	0.0564	-0.04152	0.0564	18,000	761
Toluene	16.435	0.0547611	3.285670	0.547611	0.547611	0.920080	0.2021	0.913500	0.7969	0.91350	-0.7969	0.041522	0.0564	-0.04152	0.0564	27,000	123
Ethyl Benzene	1.099225	0.8187586	49.12552	8.187586	8.187586	-7.12479	2	0.913500	0.7969	0.91350	-0.7969	0.041522	0.0564	-0.04152	0.0564	11,000	494
Phenol	1.9072	0.4718959	28.31375	4.718959	4.718959	-4.98173	2	0.913500	0.7969	0.91350	0.7969	0.041522	0.0564	-0.04152	0.0564	7,000	315
Isophorone	1.007245	0.8935264	53.61158	8.935264	8.935264	-7.50593	2	0.913500	0.7969	0.91350	0.7969	0.041522	0.0564	-0.04152	0.0564	26,000	1,169
2,4-Dimethylphenol	1.07875	0.8342989	50.05793	8.342989	8.342989	-7.20649	2	0.913500	0.7969	0.91350	0.7969	0.041522	0.0564	-0.04152	0.0564	140	6
Naphthalene	2.0836	0.4319447	25.91668	4.319447	4.319447	-4.67494	2	0.913500	0.7969	0.91350	0.7969	0.041522	0.0564	-0.04152	0.0564	620	28
Bis(2-ethylhexyl)phthalat	17010001	0.0000000	0.000003	0.000000	0.000000	6735.142	0	0.913500	0.7969	0.91350	0.7969	0.041522	0.0564	-0.04152	0.0564	7,250	0

Project: Summit National Site
Contaminant Transport Calculations for Water-Table Aquifer

By: DBT Date: 10-Dec-87

SEE TRANSPORT CALCULATIONS OF 2/26/87 FOR TERMS, EQUATIONS AND REFERENCES

HIGH-VELOCITY CASE

v= 0.9 ft/day
time= 3650 days
x= 4500 feet

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.7166860	43.00116	7.166860	7.166860	2.377861	0.0011	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	24,000	0
1,1-Dichloroethane	2.94355	0.3057532	18.34519	3.057532	3.057532	6.538721	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	12,000	0
1,2-Dichloroethane	1.9513	0.4612309	27.67385	4.612309	4.612309	4.430977	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	115,000	0
2-Butanone	0.998141	0.9016757	54.10054	9.016757	9.016757	1.360213	0.0477	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	650,000	209
1,1,1-Trichloroethane	5.662	0.1589544	9.537265	1.589544	1.589544	10.50454	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	53,000	0
Trichloroethene	7.1425	0.1260063	7.560378	1.260063	1.260063	12.16022	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	18,000	0
Toluene	16.435	0.0547611	3.285670	0.547611	0.547611	19.63325	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	27,000	0
Ethyl Benzene	1.099225	0.8187586	49.12552	8.187586	8.187586	1.784789	0.0109	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	11,000	1
Phenol	1.9072	0.4718959	28.31375	4.718959	4.718959	4.326075	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	7,000	0
Isophorone	1.007245	0.8935264	53.61158	8.935264	8.935264	1.400023	0.0047	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	26,000	1
2,4-Dimethylphenol	1.07875	0.8342989	50.05793	8.342989	8.342989	1.701739	0.0162	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	140	0
Naphthalene	2.0836	0.4319447	25.91668	4.319447	4.319447	4.752502	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	620	0
Bis(2-ethylhexyl)phthalat	17010001	0.0000000	0.000003	0.000000	0.000000	20902.16	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	7,250	0

v= 0.9 ft/day
time= 18250 days
x= 4500 feet

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.7166860	43.00116	7.166860	7.166860	-4.84240	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	24,000	324
1,1-Dichloroethane	2.94355	0.3057532	18.34519	3.057532	3.057532	-0.93325	1.7969	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	12,000	145
1,2-Dichloroethane	1.9513	0.4612309	27.67385	4.612309	4.612309	-2.75618	1.9999	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	115,000	1,550
2-Butanone	0.998141	0.9016757	54.10054	9.016757	9.016757	-6.01600	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	650,000	8,762
1,1,1-Trichloroethane	5.662	0.1589544	9.537265	1.589544	1.589544	1.916448	0.0072	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	53,000	3
Trichloroethene	7.1425	0.1260063	7.560378	1.260063	1.260063	2.961866	0.000022	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	18,000	0
Toluene	16.435	0.0547611	3.285670	0.547611	0.547611	7.147761	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	27,000	0
Ethyl Benzene	1.099225	0.8187586	49.12552	8.187586	8.187586	-5.51420	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	0.02357	-0.02589	11,000	148
Phenol	1.9072	0.4718959	28.31375	4.718959	4.718959	-2.86024	1.9999	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	7,000	94
Isophorone	1.007245	0.8935264	53.61158	8.935264	8.935264	-5.96820	2	0.518544	0.5205	0.51854	0.5205	0.023570	0.025898	-0.02357	-0.02589	26,000	350
2,4-Dimethylphenol	1.07875	0.8342989	50.05793	8.342989	8.342989	-5.61697	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	140	2
Naphthalene	2.0836	0.4319447	25.91668	4.319447	4.319447	-2.45951	1.9993	0.518544	0.5205	0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	620	8
Bis(2-ethylhexyl)phthalat	17010001	0.0000000	0.000003	0.000000	0.000000	9347.732	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	7,250	0

v= 0.9 ft/day
time= 36500 days
x= 4500 feet

CONTAMINANT	kd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.7166860	43.00116	7.166860	7.166860	-8.64415	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	24,000	324
1,1-Dichloroethane	2.94355	0.3057532	18.34519	3.057532	3.057532	-4.06945	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	12,000	162
1,2 Dichloroethane	1.9513	0.4612309	27.67385	4.612309	4.612309	-6.13656	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	115,000	1,550
2-Butanone	0.998141	0.9016757	54.10054	9.016757	9.016757	-10.1090	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	650,000	8,762
1,1,1 Trichloroethane	5.662	0.1589544	9.537265	1.589544	1.589544	-1.10323	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	53,000	714
Trichloroethene	7.1425	0.1260063	7.560378	1.260063	1.260063	-0.09444	1.1125	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	18,000	135
Toluene	16.435	0.0547611	3.285670	0.547611	0.547611	3.611294	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	27,000	0
Ethyl benzene	1.099225	0.8187586	49.12552	8.187586	8.187586	-9.47654	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	11,000	148
Phenol	1.9072	0.4718959	28.31375	4.718959	4.718959	-6.25828	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	7,000	94
Isophorone	1.007245	0.8935264	53.61158	8.935264	8.935264	-10.0487	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	26,000	350
2,4-Dimethylphenol	1.07875	0.8342989	50.05793	8.342989	8.342989	-9.59967	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	140	2
Naphthalene	2.0836	0.4319447	25.91668	4.319447	4.319447	-5.79165	2	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	620	8
Bis(2-ethylhexyl)phthalat	17010001	0.0000000	0.000003	0.000000	0.000000	6609.843	0	0.518544	0.5205	-0.51854	-0.5205	0.023570	0.025898	-0.02357	-0.02589	7,250	0

Project: Summit National Site
Contaminant Transport Calculations for Water Table Aquifer

By: DBT Date: 10-Dec-87

SEE TRANSPORT CALCULATIONS OF 2/26/87 FOR TERMS, EQUATIONS AND REFERENCES

LOW VELOCITY CASE

v= 0.03 ft/day
time= 3650 days
x= 1450 feet

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.0238895	43.00116	7.166860	7.166860	1.719953	0.0162	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	24,000	0
1,1-Dichloroethane	2.94355	0.0101917	18.34519	3.057532	3.057532	2.729877	0.0001	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	12,000	0
1,2-Dichloroethane	1.9513	0.0153743	27.67385	4.612309	4.612309	2.192881	0.0019	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	115,000	0
2-Butanone	0.998141	0.0300558	54.10054	9.016757	9.016757	1.508076	0.0339	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	650,000	15
1,1,1 Trichloroethane	5.662	0.0052984	9.537265	1.589544	1.589544	3.833965	0	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	53,000	0
Trichloroethene	7.1425	0.0042002	7.560378	1.260063	1.260063	4.318208	0	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	18,000	0
Toluene	16.435	0.0018253	3.285670	0.547611	0.547611	6.589908	0	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	27,000	0
Ethyl Benzene	1.099225	0.0272919	49.12552	8.187586	8.187586	1.594510	0.0237	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	11,000	0
Phenol	1.9072	0.0157298	28.31375	4.718959	4.718959	2.165942	0.0019	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	7,000	0
Isophorone	1.007245	0.0297842	53.61158	8.935264	8.935264	1.516059	0.0339	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	26,000	1
2,4-Dimethylphenol	1.07875	0.0278099	50.05793	8.342989	8.342989	1.577378	0.0339	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	140	0
Naphthalene	2.0836	0.0143981	25.91668	4.319447	4.319447	2.221794	0.0011	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	620	0
Bis(2-ethylhexyl)phthalat	17010001	0.0000000	0.000003	0.000000	0.000000	6735.143	0	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	7,250	0

v= 0.03 ft/day
time= 18250 days
x= 1450 feet

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.0238895	43.00116	7.166860	7.166860	0.572325	0.4367	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	24,000	7
1,1-Dichloroethane	2.94355	0.0101917	18.34519	3.057532	3.057532	1.092256	0.1198	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	12,000	1
1,2 Dichloroethane	1.9513	0.0153743	27.67385	4.612309	4.612309	0.822760	0.2579	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	115,000	20
2-Butanone	0.998141	0.0300558	54.10054	9.016757	9.016757	0.453622	0.5245	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	650,000	229
1,1,1-Trichloroethane	5.662	0.0052984	9.537265	1.589544	1.589544	1.671890	0.0237	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	53,000	1
Trichloroethene	7.1425	0.0042002	7.560378	1.260063	1.260063	1.848616	0.0169	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	18,000	0
Toluene	16.435	0.0018253	3.285670	0.547611	0.547611	2.892680	0.00041	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	27,000	0
Ethyl Benzene	1.099225	0.0272919	49.12552	8.187586	8.187586	0.502673	0.4795	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	11,000	4
Phenol	1.9072	0.0157298	28.31375	4.718959	4.718959	0.868897	0.2579	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	7,000	1
Isophorone	1.007245	0.0297842	53.61158	8.935264	8.935264	0.458191	0.5245	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	26,000	9
2,4 Dimethylphenol	1.07875	0.0278099	50.05793	8.342989	8.342989	0.493024	0.4795	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	140	0
Naphthalene	2.0836	0.0143981	25.91668	4.319447	4.319447	0.865147	0.2293	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	620	0
Bis(2 ethylhexyl)phthalat	17010001	0.0000000	0.000003	0.000000	0.000000	3612.047	0	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	7,250	0

v= 0.03 ft/day
time= 36500 days
x= 1450 feet

CONTAMINANT	kd	v'	Dx'	Dy'	Dz'	A	erf(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.0238895	43.00116	7.166860	7.166860	0.230693	0.7237	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	24,000	12
1,1-Dichloroethane	2.94355	0.0101917	18.34519	3.057532	3.057532	0.658690	0.358	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	12,000	3
1,2-Dichloroethane	1.9513	0.0153743	27.67385	4.612309	4.612309	0.442191	0.5245	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	115,000	41
2 Butanone	0.998141	0.0300558	54.10054	9.016757	9.016757	0.125588	0.832	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	650,000	363
1,1,1 Trichloroethane	5.662	0.0052984	9.537265	1.589544	1.589544	1.064904	0.1198	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	53,000	4
Trichloroethene	7.1425	0.0042002	7.560378	1.260063	1.260063	1.234209	0.0897	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	18,000	1
Toluene	16.435	0.0018253	3.285670	0.547611	0.547611	1.997335	0.0047	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	27,000	0
Ethyl Benzene	1.099225	0.0272919	49.12552	8.187586	8.187586	0.169463	0.832	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	11,000	6
Phenol	1.9672	0.0157298	28.31375	4.718959	4.718959	0.430783	0.5245	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	7,000	2
Isophorone	1.007245	0.0297842	53.61158	8.935264	8.935264	0.129703	0.832	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	26,000	15
2,4-Dimethylphenol	1.07875	0.0278099	50.05793	8.342989	8.342989	0.160883	0.832	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	140	0
Naphthalene	2.0836	0.0143981	25.91668	4.319447	4.319447	0.475253	0.4795	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	620	0
bis(2-ethylhexyl)phthalat	7010001	0.0000000	0.000003	0.000000	0.000000	2129.839	0	0.166781	0.168	-0.16678	-0.168	0.007580	0.008	-0.00758	-0.008	7,250	0

v= 0.4" ft/day
time= 16500 days
x= 4500 feet

CONTAMINANT	Kd	v'	αx'	αy'	αz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.0238895	43.00116	7.166860	7.166860	1.447953	0.0477	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	20,000	1
1,1-Dichloroethane	2.94355	0.0101917	18.34519	3.057532	3.057532	2.522331	0.0004	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	12,000	0
1,2-Dichloroethane	1.9513	0.0153743	27.67385	4.612309	4.612309	1.959551	0.0047	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	115,000	1
2-Butanone	0.998141	0.0300558	54.10054	9.016757	9.016757	1.210820	0.0897	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	650,000	76
1,1,1-Trichloroethane	5.662	0.0052984	9.537265	1.589544	1.589544	3.649612	0	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	53,000	0
Trichloroethene	7.1425	0.0042002	7.560378	1.260063	1.260063	4.137240	0	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	18,000	0
Toluene	16.435	0.0018253	3.285670	0.547611	0.547611	6.400971	0	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	27,000	0
Ethyl Benzene	1.099225	0.0272919	49.12552	8.187586	8.187586	1.308322	0.066	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	11,000	1
Phenol	1.9072	0.0157298	28.31375	4.718959	4.718959	1.930899	0.0072	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	7,000	0
Isophorone	1.007245	0.0297842	53.61158	8.935264	8.935264	1.219873	0.0897	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	26,000	3
2,4-Dimethylphenol	1.07875	0.0278099	50.05793	8.342989	8.342989	1.289086	0.066	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	140	0
Naphthalene	2.0836	0.0143981	25.91668	4.319447	4.319447	2.043208	0.0047	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	620	0
Bis(2 ethylhexyl)phthalat	3910001	0.0000000	0.000003	0.000000	0.000000	6609.846	0	0.094672	0.5205	-0.09467	-0.5205	0.004303	0.005	-0.00430	-0.005	7,250	0

Project: Somail National Site
Contaminant Transport Calculations for Water-Table Aquifer

By: DBT Date: 10-Dec-87

SEE TRANSPORT CALCULATIONS OF 2/26/87 FOR TERMS, EQUATIONS AND REFERENCES

LOW-VELOCITY CASE

v= 0.03 ft/day
time= 3650 days
x= 4500 feet

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.0238895	43.00116	7.166860	7.166860	5.569266	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	24,000	0
1,1-Dichloroethane	2.94355	0.0101917	18.34519	3.057532	3.057532	8.623227	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	12,000	0
1,2-Dichloroethane	1.9513	0.0153743	27.67385	4.612309	4.612309	6.991194	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	115,000	0
2-Butanone	0.998141	0.0300558	54.10054	9.016757	9.016757	4.939881	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	650,000	0
1,1,1-Trichloroethane	5.662	0.0052984	9.537265	1.589544	1.589544	12.00752	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	53,000	0
Trichloroethene	7.1425	0.0042002	7.560378	1.260063	1.260063	13.49839	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	18,000	0
Toluene	16.435	0.0018253	3.285670	0.547611	0.547611	20.51542	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	27,000	0
Ethyl Benzene	1.099225	0.0272919	49.12552	8.187586	8.187586	5.195897	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	11,000	0
Phenol	1.9072	0.0157298	28.31375	4.718959	4.718959	6.909723	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	7,000	0
Isophorone	1.007245	0.0297842	53.61158	8.935264	8.935264	4.963478	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	26,000	0
2,4-Dimethylphenol	1.07875	0.0278099	50.05793	8.342989	8.342989	5.145067	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	140	0
Naphthalene	2.0836	0.0143981	25.91668	4.319447	4.319447	7.230105	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	620	0
Bis(2 ethylhexyl)phthalat	17010001	0.0000000	0.000003	0.000000	0.000000	20902.16	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	7,250	0

v= 0.03 ft/day
time= 18250 days
x= 4500 feet

CONTAMINANT	Rd	v'	Dx'	Dy'	Dz'	A	erfc(A)	B	erf(B)	C	erf(C)	D	erf(D)	E	erf(E)	Co ppb	C ppb
Methylene Chloride	1.25578	0.0238895	43.00116	7.166860	7.166860	2.293791	0.0011	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	24,000	0
1,1-Dichloroethane	2.94355	0.0101917	18.34519	3.057532	3.057532	3.727842	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	12,000	0
1,2-Dichloroethane	1.9513	0.0153743	27.67385	4.612309	4.612309	2.968631	0.000041	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	-0.005	115,000	0
2-Butanone	0.998141	0.0300558	54.10054	9.016757	9.016757	1.988371	0.0047	0.094672	0.1124	0.09467	-0.1124	0.004303	0.005	-0.00430	0.005	650,000	1
1,1,1-Trichloroethane	5.662	0.0052984	9.537265	1.589544	1.589544	5.277219	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	0.005	53,000	0
Trichloroethene	7.1425	0.0042002	7.560378	1.260063	1.260063	5.954122	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	0.005	18,000	0
Toluene	16.435	0.0018253	3.285670	0.547611	0.547611	9.120361	0	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	0.00430	-0.005	27,000	0
Ethyl Benzene	1.099225	0.0272919	49.12552	8.187586	8.187586	2.113263	0.003	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	0.00430	0.005	11,000	0
Phenol	1.9072	0.0157298	28.31375	4.718959	4.718959	2.930380	0.000041	0.094672	0.1124	-0.09467	-0.1124	0.004303	0.005	-0.00430	0.005	7,000	0
Isophorone	1.007245	0.0297842	53.61158	8.935264	8.935264	1.999924	0.0047	0.094672	0.1124	0.09467	-0.1124	0.004303	0.005	0.00430	0.005	26,000	0
2,4-Dimethylphenol	1.07875	0.0278099	50.05793	8.342989	8.342989	2.088543	0.003	0.094672	0.1124	0.09467	-0.1124	0.004303	0.005	0.00430	0.005	140	0
Naphthalene	2.0836	0.0143981	25.91668	4.319447	4.319447	3.080571	0	0.094672	0.1124	0.09467	-0.1124	0.004303	0.005	0.00430	0.005	620	0
Bis(2 ethylhexyl)phthalat	17010001	0.0000000	0.000003	0.000000	0.000000	9347.734	0	0.094672	0.1124	0.09467	-0.1124	0.004303	0.005	0.00430	0.005	7,250	0

Appendix F

APPENDIX F - RADIOLOGICAL MONITORING DATA

Eberline Thermo Electron

P. O. Box 2108
Santa Fe, New Mexico 87501
(505) 471-3232 TWX 910-983-0578

CURRENT TLD OCCUPATIONAL RADIATION EXPOSURE REPORT

5617 1 /

DATE 02/06/85

DOSIMETRY SERVICES

	NAME												
0000	CONTROL		T	11/01/84		28							
			M	02/04/85		28							
0025			T	11/01/84		2	0	0	0	0	0	1.250	5
			M	02/04/85		2	0	0	0	0	0	0.000	
0026			T	11/01/84		7	0	0	0	0	0	1.250	5
			M	02/04/85		7	0	0	0	0	0	0.000	
0027			T	11/01/84		7	0	0	0	0	0	1.250	5
			M	02/04/85		7	0	0	0	0	0	0.000	
0028			T	11/01/84		11	0	11	0	11	0	1.250	5
			M	02/04/85		0	0	11	0	11	0	0.000	
0029			T	11/01/84		19	0	19	0	19	0	1.250	5
			M	02/04/85		0	0	19	0	19	0	0.000	
0030			T	11/01/84		0	0	0	0	0	0	1.250	5
			M	02/04/85		0	0	0	0	0	0	0.000	
0031			T	11/01/84		0	0	0	0	0	0	1.250	5
			M	02/04/85		5	0	0	0	0	0	0.000	
0032			T	11/01/84		2	0	0	0	0	0	1.250	5
			M	02/04/85		2	0	0	0	0	0	0.000	
0033			T	11/01/84		0	0	0	0	0	0	1.250	5
			M	02/04/85		0	0	0	0	0	0	0.000	
0034			T	11/01/84		0	0	0	0	0	0	1.250	5
			M	02/04/85		0	0	0	0	0	0	0.000	
0035			T	11/01/84		0	0	0	0	0	0	1.250	5
			M	02/04/85		0	0	0	0	0	0	0.000	

PHOENIX SAFETY ASSOC. LTD
LORI EMPEMRIEL
P.O. BOX 545
PHOENIXVILLE P. [illegible]

Data Processed 10/16/3107

Note: Any net counts listed under (Total TLD Net Counts), < 10 are recorded as 0 mrem for total dose because background readings are taken into consideration.

Badge # 0028 and 0029 have readings of 11 & 19 for total TLD Net counts which is beta + GAMMA emitted as the [illegible] dose on radiation.

EXPLANATION AND REMARKS CONCERNING THE REPORT

1. ABOUT THE REPORT

This report lists the deep and shallow dose equivalents for each participant based on the amount of radiation to which the badge or dosimeter was exposed. The premise is that the radiation which exposed the badge is closely related in both quality and quantity to that radiation which exposed that part of the body on which the dosimeter was worn.

2. USE OF CONTROL DOSIMETER

A control dosimeter is included with the shipment of dosimeters as a means to determine radiation doses received during transit. The control dosimeter reading is subtracted from the dosimeter reading of each participant. Failure to include a control dosimeter with your return shipment of dosimeters will not allow us to assess transit exposure to radiation and such transit doses will be reflected in the participant dose.

3. MINIMUM DOSE EQUIVALENT REPORTED

Dose equivalents for the current monitoring period below the minimum measurable quantity are recorded as "M". The minimum measurable quantity depends on the dosimeter type and quality of radiation. GARDRAY® film and TLD dosimeters have a minimum reporting value of 10mrem for x and gamma rays and 40mrem for energetic beta particles. DEX RAY® film dosimeters have a minimum reporting value of 5mrem. All fast and moderate energy neutron dosimeters have a minimum reporting dose of 20mrem. Film dosimeters for thermal neutrons have a minimum reporting dose of 10mrem.

4. CUMULATIVE TOTAL DATA

Cumulative totals equal the sum of the readings of dosimeters returned for processing and reported to date. Minimal exposures are added as zero. Calendar quarters are selected to most nearly conform to NRC and state recording requirements considering starting date and length of monitoring periods chosen.

5. ADJUSTMENTS TO CUMULATIVE DATA

To aid in proper presentation of information for records required by the NRC and other regulatory bodies, amendments in the cumulative totals may have been made increasing or decreasing the totals from those originally reported. Such amendments are made at the request of an authorized representative of the client and are only to reflect supplementary data demonstrating that the dosimeter exposure originally occurred in such a manner as to either overstate or understate the true dosage to the badge assignee. (Refer to adjustment column.) Addition of previous exposure prior to commencement of use of the particular dosimeter assignment reported may also be reflected in the cumulative totals.

6. DEEP AND SHALLOW DOSE EQUIVALENTS

The deep dose is the dose equivalent from all radiations at a depth of 1cm (1000mg/cm²) in soft tissue. Considered are the effects of build up and attenuation of radiation in the body as estimated using the 30cm diameter sphere of tissue equivalent material specified by the International Commission on Radiation Units and Measurements. The shallow dose equivalent is the dose equivalent from all radiations at approximately 0.007cm depth (7mg/cm²) in soft tissue. Similar to deep dose, the contribution to the shallow dose equivalent from radiation scattered within the body is considered. The deep dose should be considered equivalent to the whole body dose and the shallow dose as equivalent to the dose to the skin of the whole body.

Dose equivalents arising from exposures to x or gamma rays will have a deep and shallow value reported. Depending on the energy of the x or gamma rays, the deep and shallow values may or may not be equal. For neutron exposures, the same value is reported for the deep and shallow dose equivalent. Beta exposures are only reported as a shallow dose equivalent.

When the lens of the eyes are not sufficiently protected from radiation, NRC Form 5 states that doses at 300mg/cm² or less should be measured. When dosimeter data indicate that reporting the dose at 1cm depth would not reflect the appropriate dose at 0.3cm depth (300mg/cm²) the deep dose reported will be the dose at 0.3cm depth rather than at 1.0cm depth.

7. RING BADGE READINGS

Ring badge readings are reported as a shallow dose as if due to gamma rays. If produced by low energy x-rays or beta particles, the reported value may be incorrect. Calibration factors for x rays and beta particles are available so that a more accurate interpretation is possible.

8. GENERAL RADIATION EXPOSURE GUIDES*

TYPE OF EXPOSURE	GUIDE VALUE
Whole body, head & trunk, blood forming organs, lens of eye or gonads	1250 millirems per quarter. 5000 millirems per year. Up to 3000 millirems is permitted in a calendar quarter as long as the accumulated occupational dose to the whole body does not exceed 5000 millirems x (age 18)
Skin of whole body	7,500 millirems per quarter
Hand, forearms, feet and ankles	18,750 millirems per quarter

*U.S.N.R.C. regulations, Title 10, Part 20, Code of Federal Regulations (9-1-78). NOTE: Certain states and other regulatory agencies may follow guides that are different from the above.

COLUMN REFERENCES

NOTES (COLUMN 4)

A ABSENT

B-1 This film appears to have been damaged by light. The accuracy of any reading given would be affected thereby.

B-2 This film appears to have been damaged by moisture. The accuracy of any reading given would be affected thereby.

B-3 This film appears to have been damaged by chemical fogging. The accuracy of any reading given would be affected thereby.

B-4 This dosimeter appears to have been manufactured faultily. The accuracy of any reading given would be affected thereby.

B-5 This film appears to have been damaged by heat or pressure. The accuracy of any reading given would be affected thereby.

C Evidence of contamination.

DA This film packet appears to have been exposed out of the badge; therefore, the value given is based on a high energy gamma calibration and is valid only if the exposure were due to high energy gamma. If it were due to beta particles or lower energy x or gamma rays, the value reported may be inaccurate.

DC This film packet is partially lightstruck. There is apparently a dose recorded; however, no exact quantitative determination can be made. The reported dose is the maximum received.

DD This film badge appears to have been shielded during exposure. The dose reported is not an exact quantitative determination, but only an indication that the badge was exposed.

DE This film badge appears to have been shielded during exposure. There is a dose recorded; however, no exact quantitative determination can be made.

DF This film packet appears to have been misplaced in the badge. The dose reported is not an exact quantitative determination, but is only an indication that the badge was exposed.

DG Although this film packet was slightly lightstruck, there seems to be no apparent effect on the reading.

DH The beta-gamma background on this film badge was too high to give a valid fast neutron reading.

DI This reading is based on 50-150KV x ray. This film badge appears to be defective; please return badge for replacement.

DJ This film packet is too old to process.

DL This control packet appears to have been placed in a film badge indicating possible misuse. May we remind you that the value of the control films is lost if used for personnel monitoring or other radiation measurement purposes.

DR This film appears to have been exposed from the rear of the badge.

DS Amounts shown in columns 8 & 9 have been permanently subtracted from cumulative totals at customer's request.

DT Amounts shown in columns 8 & 9 have been permanently added to cumulative totals at customer's request.

DU Amounts shown in columns 8 & 9 have been supplied by customer for period prior to inception of Landauer service and have been permanently added to cumulative totals.

DV Amounts shown in columns 8 & 9 are previous lifetime exposures supplied by customer and have been permanently added to cumulative totals.

DY Other comment - See attached note.

DZ Other comment - See attached note.

E Irregular exposure.

E-1 Due to the irregular exposure the effective energy cannot be properly determined and the dosage is estimated arbitrarily based on 50 to 150 PKV x rays as probable source of exposure. If different energy, the value reported will not be the actual dosage.

E-2 Due to the irregular exposure the effective energy cannot be properly determined and the dosage is estimated arbitrarily based on gamma or x ray over 400KeV as probable source of exposure. If different energy, the value reported will not be the actual dosage.

H Unused.

QC Dosimeter received and processed. Quality control has not authorized reporting of doses. A separate report containing the doses for this dosimeter will be forthcoming.

DOSIMETER TYPE USED (COLUMN 6)

[illegible]

RADIATION QUALITY (COLUMN 7)

The following codes are provided to identify the types and in some cases energies of radiation contributing to the dose equivalent.

P - x or gamma ray exposure. P may be followed by an H for high energy x or gamma rays (greater than 250 keV effective), M for moderate energies (between 100 and 250 keV effective) or L for low energies (less than 100 keV effective).

B - Beta particle exposure.

N - Neutron exposure. N may be followed by an F for fast and intermediate energy neutrons or a T for thermal neutrons.

PB - Combined exposure due to both x or gamma rays and beta particles. The beta particle dose will be printed as a second line in the shallow dose column and coded as B, indicating that the second line is the beta dose component of the combined shallow dose.

PN - Combined exposure due to x or gamma rays and neutrons. The neutron dose will be printed as a second line in the deep dose column. This line will be coded as NF or NT, indicating that the second line is the dose due to fast and intermediate energy neutrons or thermal neutrons respectively.

ADJUSTMENTS (COLUMN 18)

Adjustments made to cumulative totals with this or previous report at customer request.

A - Additions
B - Subtractions
C - Additions & Subtractions
D - Dosage data supplied by customer for period prior to inception of Landauer service.
E - Dosage data supplied by customer for period prior to inception of Landauer service; additional changes have also been made.
F - Previous lifetime exposure supplied by customer.
G - Previous lifetime exposure supplied by customer; additional changes have also been made.

UNUSED PERMISSIBLE ACCUMULATED DOSE (COLUMN 17)

Unless birth date and lifetime exposure records are supplied by the customer, no values are reported in column 17. If this data is supplied, the difference between the participant's age (in years) and 18 (if age greater than 18) is multiplied by 5000. From this we subtract the value shown in column 15. The result is that value given in column 17. [illegible] January 8, 1957 recommendations of the National Committee on Radiation Protection and Measurements. These values are given for whole body exposure only.

INCEPTION DATE OF PERMANENT TOTAL (COLUMN 21)

Available to client [illegible] requirements and may not reflect actual beginning date of a [illegible] totals.

LANDAUER DOSIMETRY SERVICES ARE TESTED BY PRINCIPAL TESTING ORGANIZATIONS AGAINST CURRENT CONSENSUS INDUSTRY STANDARDS

ACCOUNT NO	CLIENT CODE
35394	

PROCESS NO	REPORT DATE	EXPOSURE THRU DATE	REPORTING TIME IN WORK DAYS	PAGE NO
T6290M	11/06/86	10/17/86	14	1

QUALITY CONTROL RELEASE
JLN

SRM ASSOCIATES INC

[redacted]

RADIATION DOSIMETRY REPORT

Summit 86-301

Landauer®

R S Landauer, Jr & Co
Division of TechOps, Inc TechOps
2 Science Road, Glenwood, Illinois 60425-1586
(312) 755-7000

Accredited by the
National Bureau of Standards
through

1 - PN 3484 - 64752

PARTICIPANT ID NUMBER	NAME	SOCIAL SECURITY NUMBER	NOTE (SEE REVERSE SIDE)	DOSIMETER TYPE USE	RADIATION QUALITY	EXPOSURE TO BADGE (MILLIREMS) FOR PERIOD(S) INDICATED BELOW		CUMULATIVE TOTALS (MILLIREMS)						ADJUSTMENTS	SHIPPED PART OF PREVIOUSLY ACCUMULATED DOSE (MILLIREMS)	SEX	BIRTH DATE			NUMBER BADGE REPORTS		RECEIPT DATE OF PERMANENT FORM	
								CALENDAR QUARTER		YEAR TO DATE		PERMANENT											
						DEEP	SHALLOW	DEEP	SHALLOW	DEEP	SHALLOW	DEEP	SHALLOW				MO	DA	YR	TO DATE	QTR	MO	YR
	FOR EXPOSURE PERIOD 06/25/86 TO 07/24/86							SECOND		1986													
*****	NO CONTROL DOSIMETER RETURNED WITH THE FOLLOWING PERSONNEL DOSIMETERS.																						
00029				T	P	30	30	30	30	30	30	30	30							1	1	5	86
00031				T	P	20	20	20	20	20	20	20	20							1	1	5	86
00032				T	P	20	20	20	20	20	20	20	20							1	1	5	86
00034				T	P	20	20	20	20	20	20	20	20							1	1	5	86
00035				T	P	30	30	30	30	30	30	30	30							1	1	5	86
00039				T	P	20	20	20	20	20	20	20	20							1	1	5	86
	FOR EXPOSURE PERIOD 06/25/86 TO 07/24/86							SECOND		1986													
			X	T	P	30	30																
00030				T	P	30	30	30	30	30	30	30	30							1	1	5	86

(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (11) (12) (13) (14) (15) (16) (17) (18) (19) (20) (21)

SRW ASSOCIATES INC
[redacted]

ACCOUNT NO	SERIES CODE
35394	

PROCESS NO	REPORT DATE	DOSIMETER RECEIVED	REPORTING TIME IN WORK DAYS	PAGE NO
T62861	10/22/86	10/13/86	7	1

QUALITY CONTROL RELEASE
JLN

Landauer®

R. S. Landauer, Jr. & Co.
Division of Tech/Ops, Inc. Tech/Ops
2 Science Road, Glenwood, Illinois 60425-1586
(312) 755-7000

Accredited by the National Bureau of Standards through

RADIATION DOSIMETRY REPORT

SUMMIT 86301

1 - PR 347[illegible] - 63412

PARTICIPANT ID NUMBER	NAME	SOCIAL SECURITY NUMBER	NOTE (SEE REVERSE SIDE)	DOSIMETER TYPE	USE	RADIATION QUALITY	EXPOSURE TO BADGE (MILLIREMS) FOR PERIOD(S) INDICATED BELOW: DEEP	SHALLOW	CUMULATIVE TOTALS (MILLIREMS) CALENDAR QUARTER: DEEP	SHALLOW	YEAR TO DATE: DEEP	SHALLOW	PERMANENT: DEEP	SHALLOW	ADJUSTMENTS	UNUSED PART OF PERMISSIBLE ACCUMULATED DOSE (MILLIREMS)	SEX	BIRTH DATE: MO	DA	YR	NUMBER BADGE REPORTS: TO DATE	QTR	INCEPTION DATE OF PERMANENT TOTAL: MO	YR
①	②	③	④	⑤	⑥	⑦	⑧	⑨	⑩	⑪	⑫	⑬	⑭	⑮	⑯	⑰	⑱	⑲			⑳		㉑	
	FOR EXPOSURE PERIOD 06/25/86 TO 07/24/86 SECOND 1986																							
*****	NO CONTROL DOSIMETER RETURNED WITH THE FOLLOWING PERSONNEL DOSIMETERS.																							
00028				1	1	P	20	20	20	20	20	20	20	20							1	1	[illegible]	

Skal ASSOCIATES, INC.

[redacted]

RADIATION DOSIMETRY REPORT

Landauer®

R S Landauer, Jr & Co
Division of TechOps Inc TechOps
2 Science Road, Glenwood, Illinois 60425-1586
(312) 755-7600

Accredited by the
National Bureau of Standards
through

PARTICIPANT ID NUMBER	NAME	SOCIAL SECURITY NUMBER	NOTE FREE (REVERSE SIDE)	DOSIMETER TYPE	USE	RADIATION QUALITY	EXPOSURE TO BADGE (MILLIREMS) FOR PERIOD(S) INDICATED BELOW DEEP	EXPOSURE TO BADGE (MILLIREMS) FOR PERIOD(S) INDICATED BELOW SHALLOW	CUMULATIVE TOTALS (MILLIREMS) CALENDAR QUARTER DEEP	CALENDAR QUARTER SHALLOW	YEAR TO DATE DEEP	YEAR TO DATE SHALLOW	PERMANENT DEEP	PERMANENT SHALLOW	ADJUSTMENTS	DOSAGE PART OF PLUTONIUM ACCUMULATED DOSE (MILLIREMS)	SEX	BIRTH DATE MO	BIRTH DATE DA	BIRTH DATE YR	NUMBER BADGE REPORTS TO DATE	NUMBER BADGE REPORTS QTR	RECEPTION DATE OF PERSONNEL MONITORING TOTAL MO	RECEPTION DATE OF PERSONNEL MONITORING TOTAL YR
	FOR EXPOSURE PERIOD ENDING																							
00000	TLD CONTROL							3																
90000									SUMMIT Bio 301															
90001																								
90002																								
90003																								
90004																								
90005																								
90006									Old Springfield Bio 245															
90007																								
90008																								
90009																								
90010																								
90011																								

(1) (2) (3) (4) (5a) (5b) (6) (7) (8) (9) (10) (11) (12) (13) (14) (15) (16) (17) (18) (19) (20) (21)

FEB 5 1996

SHM ASSOCIATES INC

[redacted]

Landauer

R S Landauer, Jr. & Co.
Division of TechOps, Inc. [redacted]
2 Science Road, Glenwood, Illinois 60425-1586
(312) 755-7000

Accredited by the
National Bureau of Standards
through [illegible]

RADIATION DOSIMETRY REPORT

Summit 12/85 - 1/86

PARTICIPANT ID NUMBER	NAME	SOCIAL SECURITY NUMBER	NOTE (SEE INSTRUCTIONS)	BADGE TYPE	LOT#	RADIATION QUALITY	EXPOSURE TO BADGE (MILLIREMS) FOR PERIOD(S) INDICATED BELOW: DEEP	EXPOSURE TO BADGE (MILLIREMS) FOR PERIOD(S) INDICATED BELOW: SHALLOW	CUMULATIVE TOTALS (MILLIREMS): CALENDAR QUARTER DEEP	CALENDAR QUARTER SHALLOW	YEAR TO DATE DEEP	YEAR TO DATE SHALLOW	PERMANENT DEEP	PERMANENT SHALLOW	ADJUSTMENTS	DOSE(S) NOT TO BE PERMANENTLY ACCUMULATED (MILLIREMS)	INDEX	BIRTH DATE MO	BIRTH DATE DA	BIRTH DATE YR	NUMBER BADGE REPORTS TO DATE	NUMBER BADGE REPORTS THIS MO	MO	YR
	FOR EXPOSURE PERIOD 11/25/85																							
00000	TLD CONTROL							M					NUTUAL											
04110	Brown, E						M	M					NUTUAL											
04111	Folenik, A						M	M					NUTUAL											
04112	Thompson, D						M	M					NUTUAL											
04113	McCracken, K						M	M					NUTUAL											
04114	Dean, P						M	M					NUTUAL											
04115	Marlin, P						M	M					NUTUAL											
04116	Barile, T						M	M					NUTUAL											
04117	Lipovich, M						M	M					NUTUAL											
04118	Lipovich, M						M	M					NUTUAL											
04119	Fisher, C						M	M					NUTUAL											
04120	Gallihue, B						M	M					NUTUAL											
04121	Harris, T						M	M					NUTUAL											
04123	Guthrie, D						M	M					NUTUAL											
04125	Harris, D						M	M					NUTUAL											
04126	Malcomb						M	M					NUTUAL											
04127							M	M					NUTUAL											
04128							M	M					NUTUAL											
04129							M	M					NUTUAL											

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21

FEB 18 1986

ACCOUNT NO	NUMBER CODE
35394	

SAIC ASSOCIATES INC
[redacted]

PROCESS NO	REPORT DATE	DOSIMETER RETURNED	REPORTING TYPE (In Work Days)	PAGE NO
04234	2/14/86	2/7/86	5	1

QUALITY CONTROL RELEASE
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RADIATION DOSIMETRY REPORT

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National Bureau of Standards
through [illegible]

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PARTICIPANT BADGE NUMBER	NAME	SOCIAL SECURITY NUMBER	NOTE (SEE REVERSE SIDE)	DOSIMETER TYPE	SEX	RADIATION QUALITY	EXPOSURE TO BADGE (MILLIREMS) FOR PERIOD(S) INDICATED BELOW		CUMULATIVE TOTALS (MILLIREMS)						ADJUSTMENTS	ANNUAL LIMIT OF PERMISSIBLE ACCUMULATION (mrem)	REF	BIRTH DATE			NUMBER BADGE REPORTS		INCEPTION DATE OF PERSONNEL MONITORING	
									CALENDAR QUARTER		YEAR TO DATE		PERMANENT					MO	DA	YR	TO DATE	DTR	MO	YR
							DEEP	SHALLOW	DEEP	SHALLOW	DEEP	SHALLOW	DEEP	SHALLOW										
	FOR EXPOSURE PERIOD 11/25/85																							
AAAAA	NO CONTROL DOSIMETER RETURNED WITH THE FOLLOWING PERSONNEL DOSIMETERS.																							
04122				B	P		20	20					NOTOTAL											
04124				B	P		20	20					NOTOTAL											

(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (11) (12) (13) (14) (15) (16) (17) (18) (19) (20) (21)

Appendix G

APPENDIX G - WESTON GEOPHYSICAL SURVEY

Weston Geophysical
CORPORATION

May 28, 1985
WGC R588-92

SRW ASSOCIATES INC.
[redacted]

Dear [redacted]:

In accordance with your authorization, a geophysical survey program was conducted at the Summit National Site in Deerfield Township, Ohio.

Preliminary data and a draft report have been previously submitted.

This submission is the final report presenting the results and findings of the geophysical investigation.

Very truly yours,

WESTON GEOPHYSICAL CORPORATION

Paul Fisk

Paul Fisk

PF/rf-2009R2

Post Office Box 550 • Westboro, Massachusetts 01581 • (617) 366-9191 • Telex No. 920491

GEOPHYSICAL SURVEY

SUMMIT NATIONAL SITE
DEERFIELD TOWNSHIP
PORTAGE COUNTY, OHIO

Prepared for

SRW ASSOCIATES, INC.

MAY 1985

Weston Geophysical
CORPORATION

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LIST OF FIGURES

1.0 EXECUTIVE SUMMARY

A multiple-technique geophysical survey was conducted at the Summit National Site in Deerfield Township, Portage County, Ohio to evaluate overall site conditions.

Magnetic data obtained along a 25-foot grid within the site identified seven possible waste container burial sites. Ground penetrating radar [GPR] surveying had limited penetration due to highly conductive surface materials. GPR test lines identified buried point targets, possibly barrels, within two of the magnetic anomalies [anomalous zones IV and VI].

Electromagnetic surveying along selected profile lines, both within and adjacent to the site, indicate higher conductivity values associated with near surface materials and the upper aquifer and lower conductivity values associated with an aquitard at depth. The high conductivity values indicative of contaminants within the upper aquifer appear to be migrating eastward more or less along the path of a stream. Lower conductivity values at depth indicate that the aquitard is slowing the vertical migration of contaminants. Contoured conductivity values indicate an eastward migration of contaminants along the top or possibly within the upper portion of the aquitard.

2.0 INTRODUCTION & PURPOSE

A geophysical survey was conducted for SRW Associates, Inc., at the U.S. EPA Summit National Site located in Deerfield Township, Portage County, Ohio during the period of October 31 through November 7, 1984. The multiple-technique geophysical program consisted of magnetometer, electromagnetic terrain conductivity [EM], ground penetrating radar [GPR] and borehole logging surveys.

The general purpose of the multiple-technique survey was to evaluate the overall site condition in terms of total field magnetics, surface and subsurface conductivity and stratigraphy. The specific objective

of the magnetometer survey was to determine those locations within the site where waste containers, drums, barrels, tanks, were buried. One of the specific objectives of the EM survey was to define a relationship between the general site stratigraphy and conductivity. The general site stratigraphy consists of surface soils, upper aquifer, aquitard and lower aquifer. The other objective of the EM survey was to evaluate the extent of groundwater contamination and the direction of contaminant migration. The specific objective of the GPR survey was to evaluate the source of the magnetic anomalies. The borehole logging survey was conducted to define various parameters: resistivty, conductivity, porosity, temperature, self potential, density and temperature related to the stratigraphy.

3.0 LOCATION & SURVEY CONTROL

The area of investigation is shown on Figure 1, a segment of the Deerfield, Ohio United States Geological Survey Quadrangle Map. The specific locations of magnetic, EM and GPR survey lines are shown on Figures 2 and 2A. The base map and horizontal control for this survey were provided by SRW Associates, Inc.

4.0 METHOD OF INVESTIGATION

The geophysical field program utilized magnetic, electromagnetic terrain conductivity and ground penetrating radar and borehole logging techniques. The method of investigation for each technique is described is the following sections.

4.1 Magnetometer Survey

Total field magnetic measurements were made using a Geometrics model G-816 Portable Proton Magnetometer. Total field magnetic readings were recorded every 10 feet on a 25-foot survey grid in the on-site area. Base station data used to correct the survey data for local diurnal variation was obtained on Line F at Station 6+75. The locations of metal debris scattered throughout the survey area were noted in field

data books, since this debris will effect magnetic measurements. A discussion of the magnetic survey technique is included as Appendix A to this report.

4.2 Electromagnetic Terrain Conductivity [EM] Survey

Conductivity measurements were made on each survey line using both the EM31 and EM34-3. Measurements were made at 12.5-foot intervals with the EM31. The data intervals for the EM34-3 survey were 25-foot and 50-foot, respectively, for the 10 and 20 meter coil spacings. A complete set of data showing the variation of conductivity with depth was obtained by making multiple traverses over each survey line. The EM31 provided information on the conductivity of the near surface material. EM34-3 data obtained with the 10- and 20-meter coil spacing, vertical coil configuration, provided information on subsurface conductivity with increasing depth. The EM34-3 20-meter coil spacing, horizontal coil configuration, provided the maximum depth of penetration achieved during this survey. The EM34-3 40-meter coil spacing, vertical coil configuration, survey originally proposed for this site could not be accomplished due to high levels of electromagnetic background noise. The 20-meter coil spacing, horizontal coil configuration, survey was used to complete the data set.

Table 1 summarizes the exploration depths of the EM31 and EM34-3 for the various coil spacings and configurations used for the survey.

TABLE 1

Type	Intercoil Spacing [meters]	Approximate Exploration Depth [feet] Vertical Coils	Horizontal Coils
EM31	3.7	-	8 - 18
EM34-3	10	20 - 25	40 - 50
EM34-3	20	40 - 50	90 - 100

A discussion of the electromagnetic conductivity technique is included as Appendix B to this report.

4.3 Ground Penetrating Radar [GPR] Survey

GPR data were obtained using a Geophysical Survey Systems, Inc. Subsurface Interface Radar [SIR] System coupled with a 300 MHz transducer. Results of the earlier EM31 survey showed high surface conductivity indicating the GPR depth of penetration would be limited. However, a test program was conducted in several areas of the site. These test lines were located in areas of magnetic anomalies and other areas of primary interest. A discussion of the ground penetrating radar survey technique is included as Appendix C to this report.

4.4 Borehole Logging Survey

Spontaneous Potential [SP], resistivity, Natural Gamma Ray, Neutron-Neutron, Density, Caliper, and Fluid temperature and conductivity geophysical logging techniques were used in borings MW-8 and MW-14.

5.0 DISCUSSION OF RESULTS

5.1 On-Site Area

5.1.1 Magnetometer Survey

Contoured magnetometer survey data [Figure 3] obtained in the on-site area defined several anomalous zones. These zones are indicated as I-VII on the Magnetic Anomaly Map [Figure 4]. Metal debris observed on the surface was noted in the field records and has been accounted for in the interpretation of magnetic data described below.

Zone I is a relatively broad area centered near the intersection of Lines H+50 and 8+50. The anomalous zone consists of several magnetic highs and lows in an area approximately 125 x 125 feet. The amplitude, 800+ gammas, of the anomaly and extent of this zone indicate a relatively large amount of buried magnetic material scattered throughout

this area. The type of material suspected to be buried is probably similar to that observed at the surface; drums, barrels, cans and general debris.

Zone II is a relatively small area centered near the intersection of Lines G+75 and 9+50. The location, size and amplitude of this magnetic anomaly appears to confirm the suspicion of a buried tank in this area, an area which coincides with seepage of a tar like material from a subsurface source.

Zone III is a relatively small area centered near the intersection of Lines F+50 and 9+00. The low amplitude, approximately 250 gammas, signature of this anomaly is indicative of a relatively small amount of magnetic material. The anomaly is probably the result of magnetic debris, barrel covers and rings, observed on the surface at this location rather than a concentration of buried material such as barrels or drums. However, there is a possibility of buried material at this location.

Zone IV is a broad area, approximately 100 x 100 feet, centered near the intersection of Lines E+30 and 9+30. Two anomalies consisting of paired magnetic highs and lows were detected in this area. The measured amplitude of the anomaly on the west side of the zone is greater than 2000 gammas, indicative of a large amounts of magnetic material concentrated in a small area, possibly a nest of buried barrels. The source of the anomaly appears to be on Line E near Station 9+30. The magnetic field at this station was very erratic and resulted in highly anomalous, nonrepeatable, data.

The anomaly on the east side of the zone has a measured amplitude of approximately 800 gammas. Although smaller in measured amplitude, the magnetic signature is indicative of a concentration of material in a small area. The location of the source of this anomaly is probably defined by the magnetic high.

The anomaly at Zone V consisted of a paired high/low magnetic signature of relatively low amplitude, approximately 500 gammas, covering a small area centered near the intersection of Lines D and 8+00. Magnetic material including barrels, steel cable and general debris was observed on the ground surface in this area. The magnetic material observed in this area contributes to the anomalous magnetic condition, however, it is considered probable that more magnetic material is present, buried in the mounds of dirt and debris at this location.

The anomalous condition in Zone VI, centered near Lines F+50 and 7+00, consisted of a low amplitude, approximately 350 gammas, magnetic high. The source of the anomaly appears to be an isolated buried magnetic object located by ground penetrating radar on Line 6+50 at F+78. The depth of burial, based on GPR data, is estimated to be two feet.

The anomalous condition in Zone VII, centered near Lines H+75 and 6+50, consisted of a paired high/low magnetic signature with an amplitude of approximately 300 gammas. Although some steel debris was observed in this area, it is considered likely that the anomaly is due to magnetic material buried at this location.

5.1.2 EM Survey

The data obtained during the electromagnetic terrain conductivity survey in the on-site area are shown as composite plots [Figures 5 and 6]. Data obtained on Line 9+00 indicates conductive surface and subsurface conditions. A zone of high conductivity was detected from approximately Line D+50 to Line G with the highest values occurring near Line F. High conductivity values, particularly near Line F, were measured with all the EM coil configurations and coil spacing utilized on this site. This conductive zone generally coincides with the magnetic anomalies, Zones III and IV, previously discussed. The data indicates a highly conductive subsurface condition extending to depth. On Line F, the EM31 data, indicates conductive subsurface condition. The anomalous, low conductivity values measured on Line F, with the 20-meter coil

spacing horizontal coil configuration, are probably the result of orientation of the instrumentation with respect to the conductive zone rather than the presence of a true low conductivity subsurface condition in this area.

Anomalous EM data were obtained on Line 7+00 at D+75. This location is adjacent to the reinforced concrete coal tipple structure. The anomalous EM condition is probably related to the influence of this structure.

In addition to the specific anomalous conditions previously discussed, several general observations of conditions in the on-site area are important. The composite plots of EM data obtained with the various coil spacings and configuration indicate that terrain conductivity generally high, 40 to 60 mmhos/m for the near surface material, decreasing to less than 40 mmhos/m at depth. The lowest conductivity values were measured with the EM34-3, 20-meter coil spacing, horizontal coil configuration. This configuration and coil spacing provided the maximum depth of penetration achieved for this survey. The composite data plots also indicate a similarity in the trend of the data obtained with the EM31 and EM34-3 [10- and 20-meter coil spacing, vertical coil configuration]. The EM31 data show the conductive nature of the near-surface materials. The EM34-3 data show relatively high conductivity conditions indicative of contamination extending to a depth which includes the upper aquifer. Due to the variation in conductivity across the site and the limited coverage, contaminant migration pathways could not be identified. Data obtained with the EM34-3, 20-meter spacing horizontal coil configuration, indicates the materials at greater depth, within the aquitard, have lower conductivity than that of the near surface material. In addition, the variation in conditions in the aquitard vary somewhat from the trends established in the shallower data. This may indicate that vertical migration of contaminants in the upper layers is variable or variation in the depth or stratigraphy of the aquitard layer.

5.1.3 GPR Survey

Due to the conductive nature of the surface materials at this site, the depth of penetration achieved by the GPR on test lines conducted in several areas was limited to approximately 2 to 3 feet. For this reason the GPR survey was determined to be inapproproate for this site. However, GPR test data did locate shallow point targets, possibly barrels, in magnetic anomaly zones IV and VI. The location of the test lines and anomalous conditions are shown on Figure 2A. Test lines conducted in the area of the scale house showed a large buried tank adjacent to the scale house on the west side and two smaller tanks buried side by side adjacent to the scale house on the east side. The depth to the top of these tanks is estimated to be less than three feet.

GPR test lines in the vicinity of the coal tipple ruins, incinerator and incinerator stacks were run to determine if tanks were buried at these locations. Several small shallow targets were detected at these locations, but signal penetration was very limited. Buried tanks, if present, were not detected within the depth range of the GPR.

5.2 Off-Site Area

5.2.1 EM Survey

The off-site electromagnetic terrain conductivity survey was conducted in an area approximately 300 by 400 feet located directly east of the on-site area. The survey coverage was established to investigate surface and subsurface conductivity in the vicinity of a small swampy area and an eastward flowing stream.

The results of the off-site EM survey are shown as data plots on Figures 6 and 7 and contour maps, Figures 8, 9, 10, and 11. Composite plots of EM data obtained with the various equipment configurations are shown for Line B and the extension of Line J+75 on Figures 6 and 7, respectively. The order in which the contour maps are presented is

consistent with increasing depth of penetration for the various EM equipment configurations. Figure 8 shows the near surface conditions, while Figure 11 shows conditions at the maximum depth of penetration. In general, this set of data shows that conductivity decreases with depth and significant variations in contour trends for the various coil spacings and configurations.

The EM31 data indicate relatively conductive, 25 to 50 mmhos/m, surface soils. The highest values were measured in the swampy area and in the vicinity of the stream channel. Surface soil conductivity values are high in the west decreasing to the north, south and east. Although the near surface soil conductivity decreases to the east, no specific trend related to the eastward flowing stream was evident in the contours. The EM34-3 data obtained with the 10- and 20-meter coil spacing, vertical coil configuration, are indicative of the conductivity in the upper aquifer. Available geologic information indicates an upper aquifer in the overall site area at a depth of approximately 22 to 30 feet. The contour maps [Figure 10 and 11] developed from 10 and 20 meter spacings, vertical coil, data indicate subsurface conductivity is relatively high, 25 to 40 mmhos/m. The measured values are high in the west, decreasing to the north, south and east. This general trend is the same as that established by the EM31 data for the near surface soils. However, unlike the near surface data, the deeper penetration EM34-3 data defined a trend evident in the contours, which may indicate a concentration of relatively high conductivity in the upper aquifer following the surface path of the eastward flowing stream. At several locations along Line 7+00, the upper aquifer conductivity is higher than the values measured for the near surface soils.

Data obtained with EM34-3 20-meter coil spacing, horizontal coil configuration, are indicative of the subsurface condition at the maximum depth of penetration achieved during the survey. These data indicate lower conductivity at depth. This decrease in conductivity with depth is probably the result of the influence of the material below the upper aquifer. Available geologic information indicates a low permeability layer, identified as an aquitard, in the site area at

a depth of 30 to 95 feet. Due to the low permeability of this layer, the vertical and horizontal migration rate of contaminants into and through the layer would be expected to be very low. However, the contours of the deep EM34-3 data, Figure 11, show conductivity values decreasing from west to east, indicating a possible migration of conductive contaminants in this layer or that the depth to the aquitard is shallower to the east. There is no indication in these data of any specific trend associated with the eastward flowing stream as was the case in the upper aquifer.

5.2.2 Borehole Logging

No direct correlation can be drawn between the stratigraphic sections and surface electromagnetic measurements because boring MW-14 is several hundred feet from the nearest EM survey line and MW-8 is located near electromagnetic Line B whose measured condutivity profile is generally characterless. These boring logs may have information which will contribute to the overall project and have been included as Figures 12 and 13.

6.0 CONCLUSIONS & RECOMMENDATIONS

The multiple-technique geophysical survey conducted for SRW Associates, Inc. at the Summit National Site obtained magnetic, electromagnetic, ground penetrating radar and borehole logging data to evaluate the overall site conditions. Geophysical data were obtained on 25-foot spaced grid lines within the on-site area and along selected survey lines in the off-site area. The 25-foot magnetic data acquisition grid provided sufficient data density to locate specific areas where barrels may be burried; and the wider spacing of EM survey lines is cost effective in evaluating the overall site conductivity conditions.

Magnetic survey results indicate seven anomalous zones where waste containers may be buried. Point targets, possibly barrels, were identified within two of the anomalous zones by the GPR survey. The electromagnetic survey results indicate that the surface materials and

upper aquifer are more conductive than the aquitard at depth. Contoured conductivity values indicate that contaminants in the upper aquifer are following the surface path of an eastwardly flowing stream and that the aquitard is limiting the vertical migration of contaminants.

It is recommended that the results of this geophysical investigation be verified with trenching of magnetic anomalies and drilling and sample analysis of possibly contaminated ground water along the migration path identified by contoured conductivity data [EM results]. It is also recommended that the geophysical survey be extended to include all of the geophysically logged boreholes to allow for direct correlation and a better overall understanding of site conditions.

FIGURES

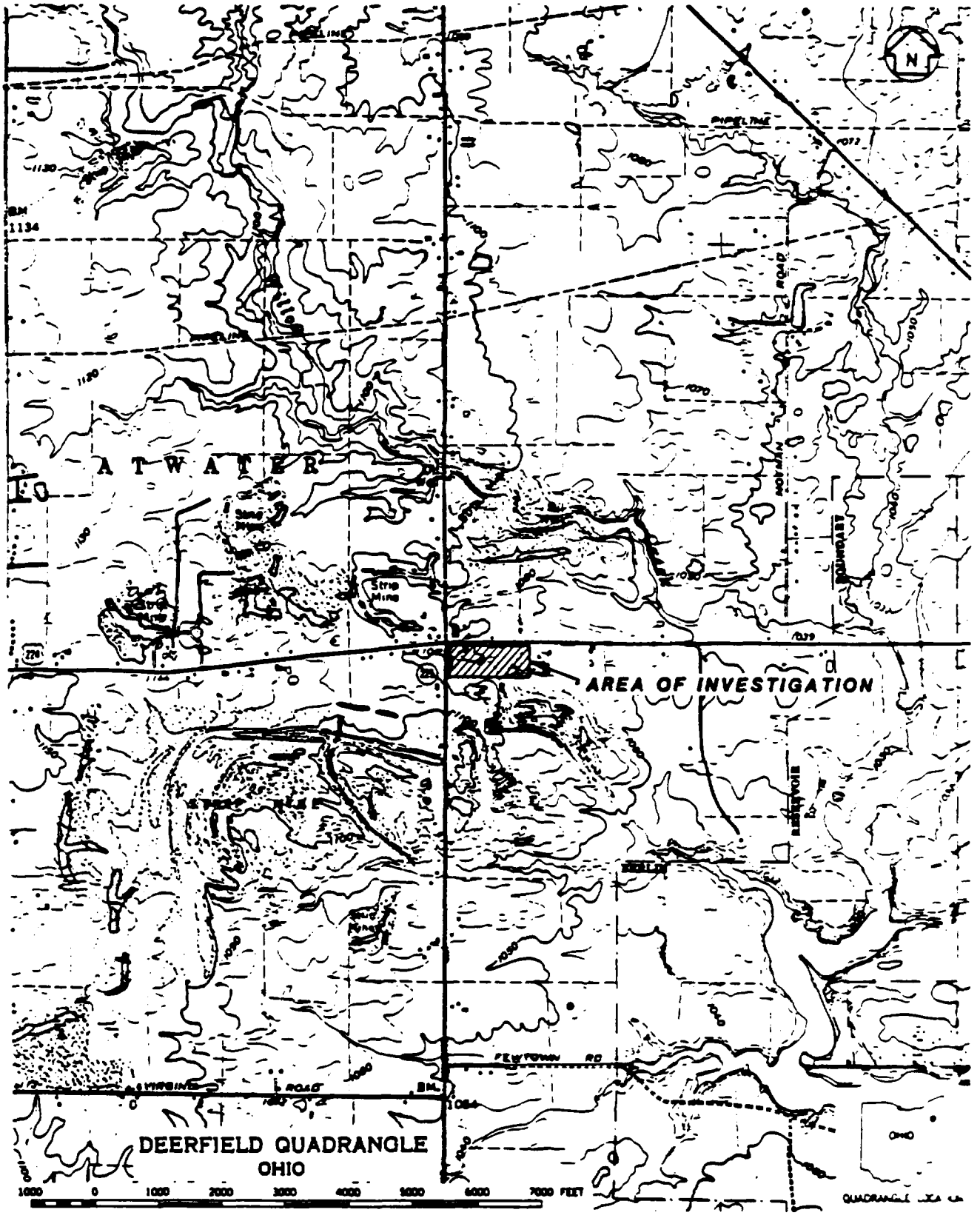


FIGURE 1
AREA OF INVESTIGATION
SUMMIT NATIONAL SITE

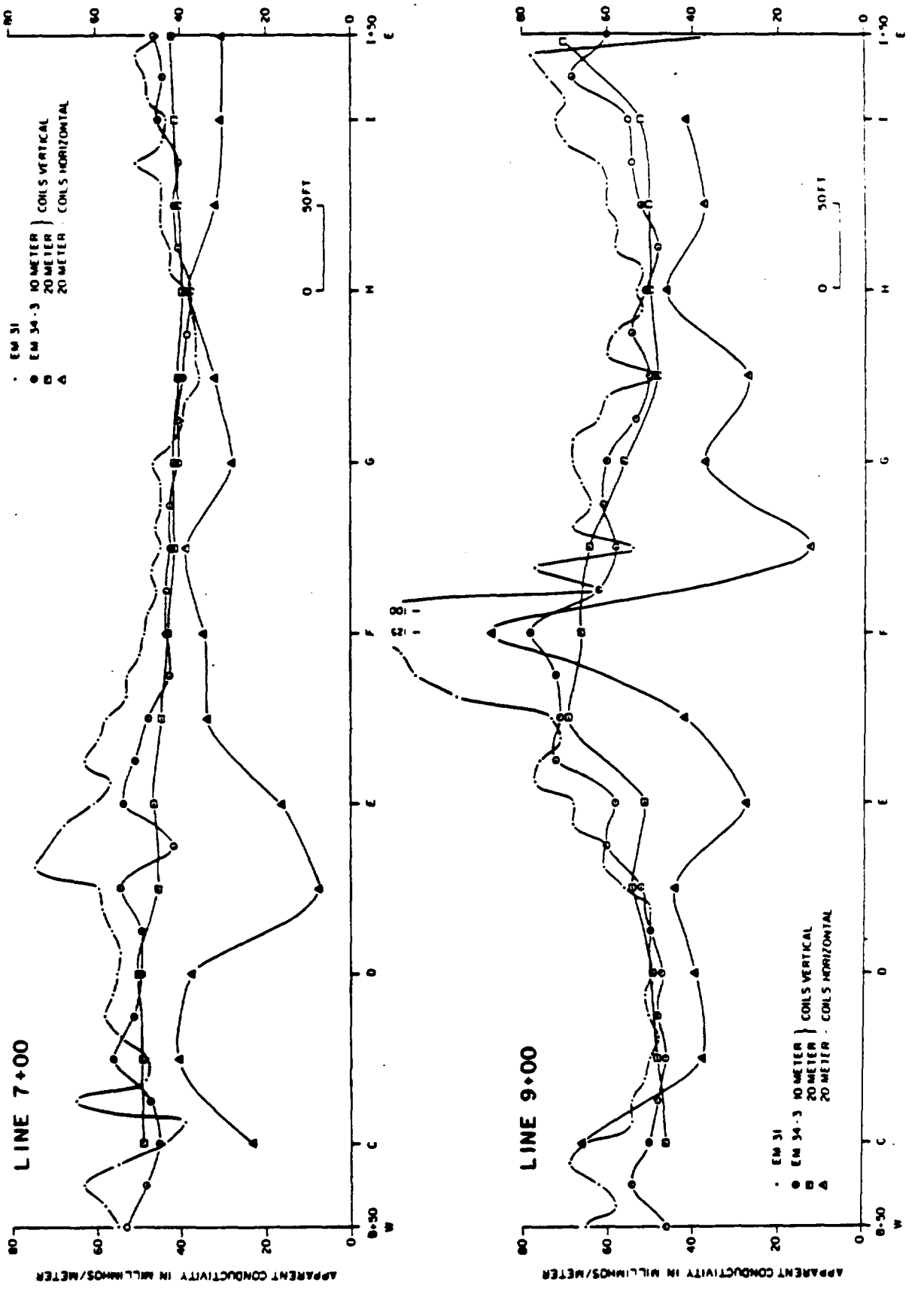
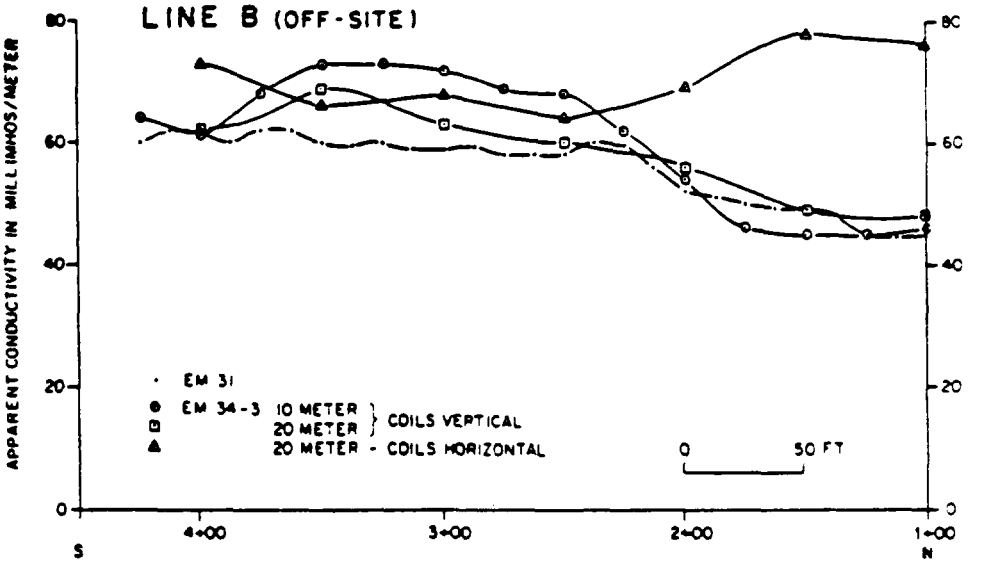


FIGURE 5
TERRAIN CONDUCTIVITY DATA
LINES 7+00 AND 9+00 (ON-SITE)
SUMMIT NATIONAL SITE



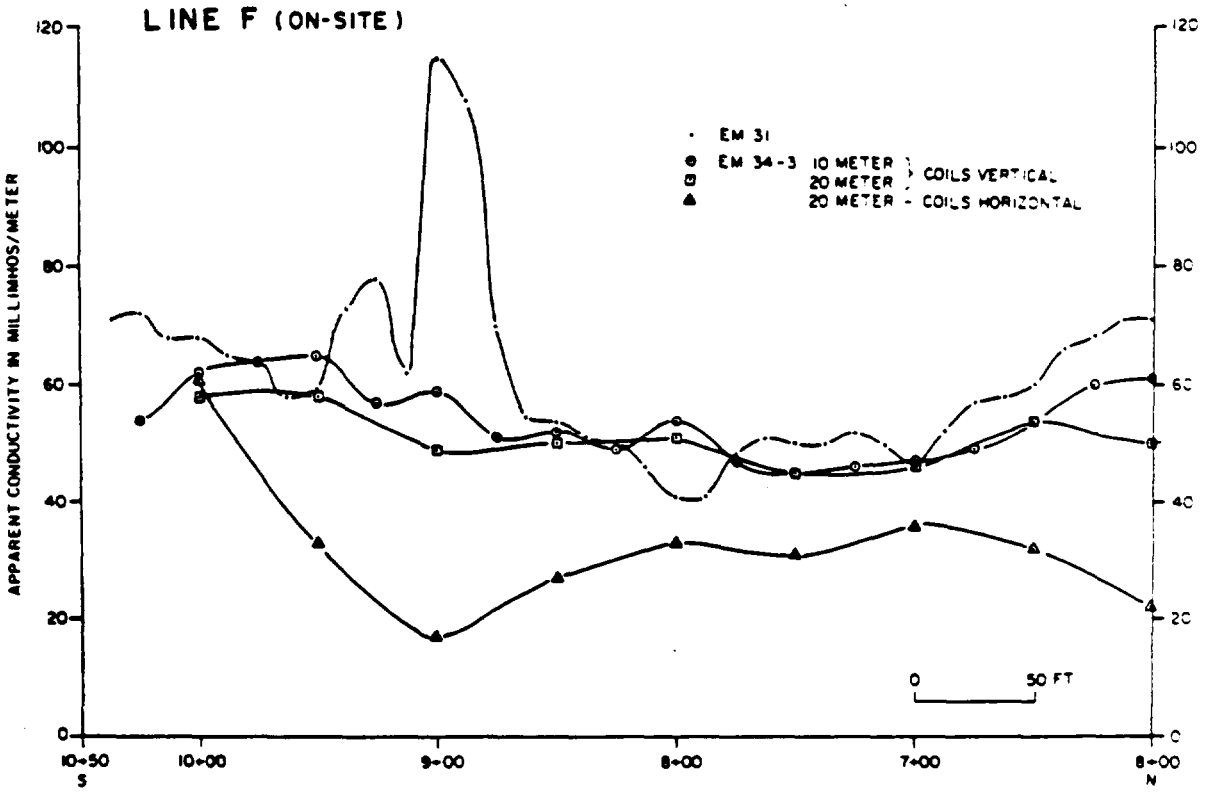


FIGURE 6
TERRAIN CONDUCTIVITY DATA
LINES F AND B (ON/OFF-SITE)
SUMMIT NATIONAL SITE

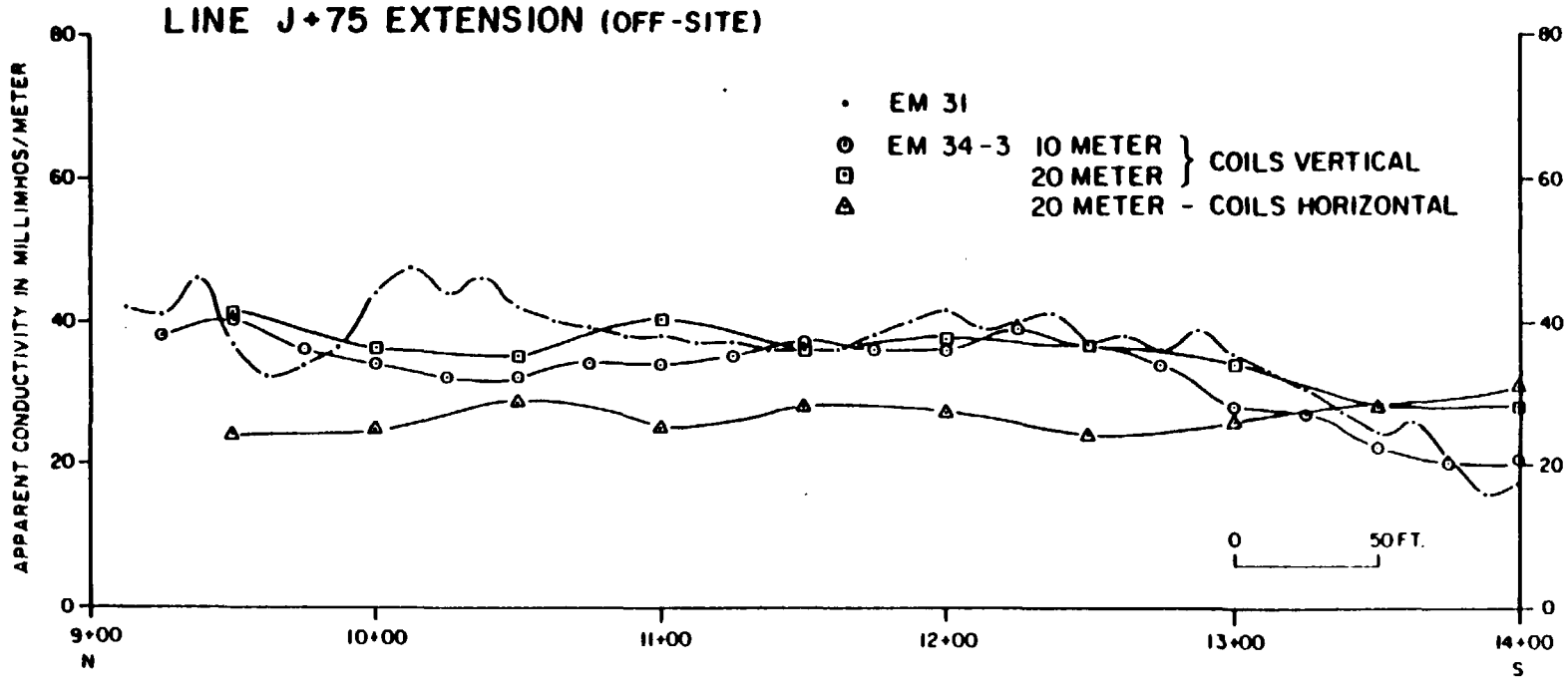
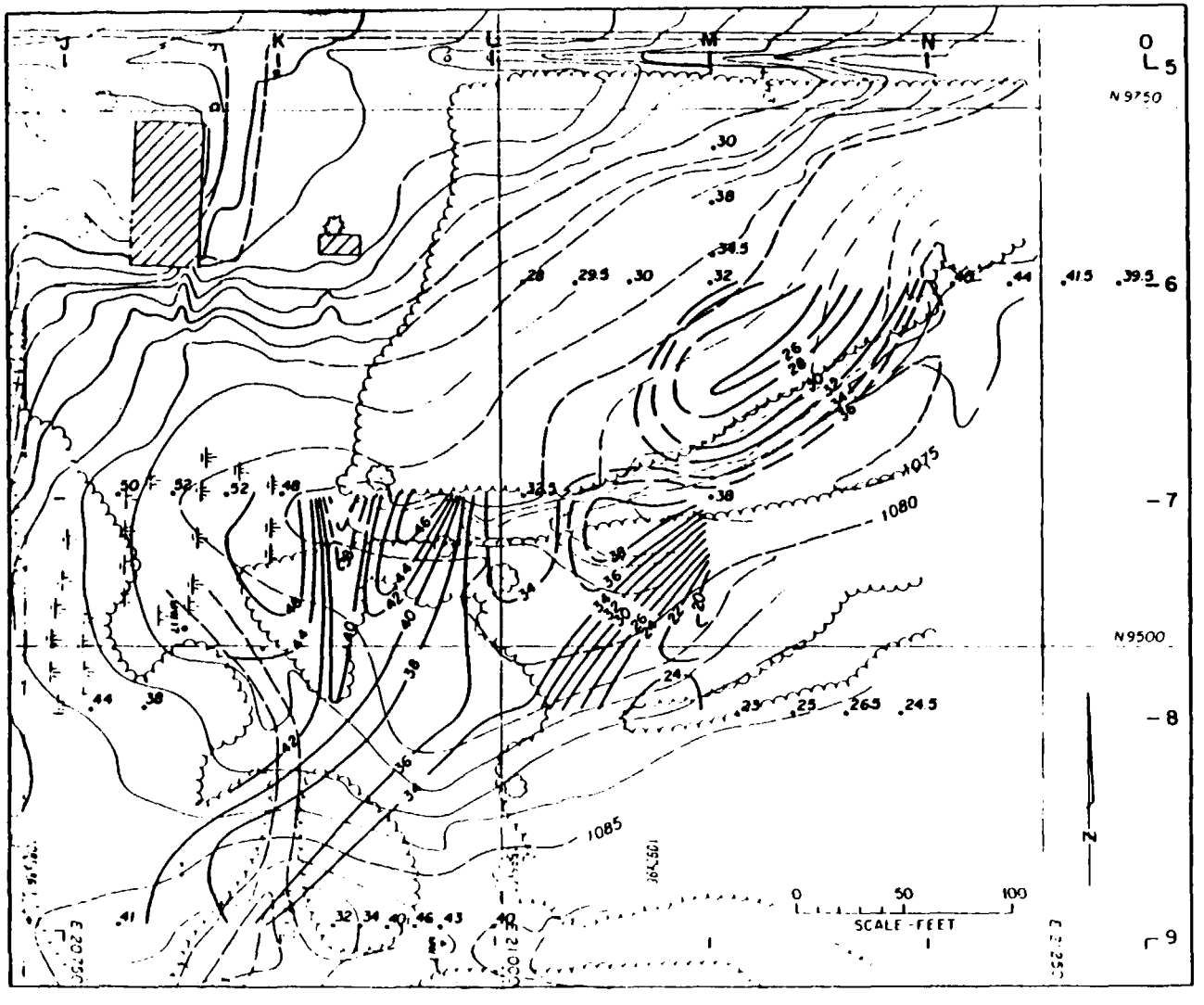


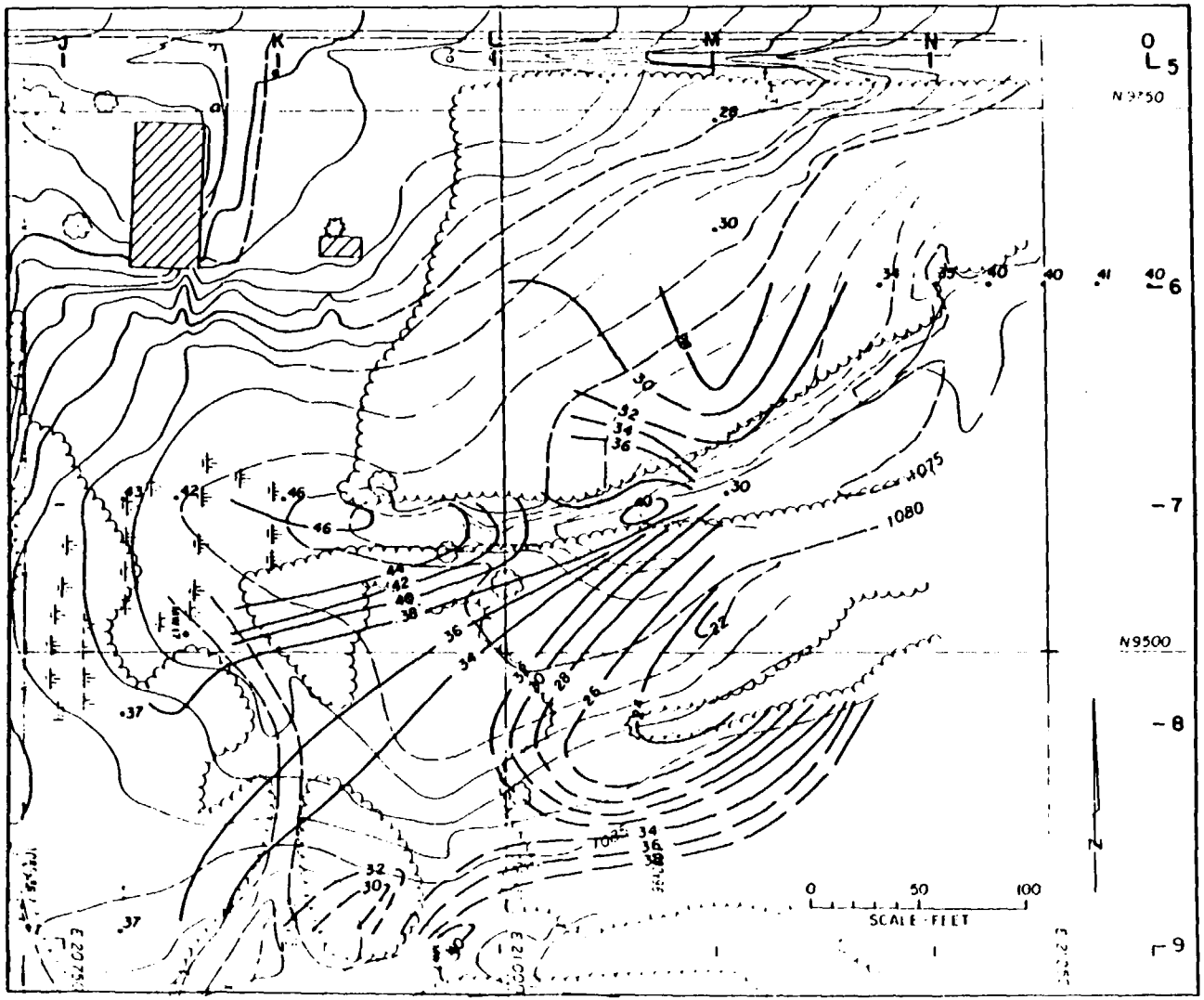
FIGURE 7
TERRAIN CONDUCTIVITY DATA
LINE J+75 EXTENSION (OFF-SITE)
SUMMIT NATIONAL SITE



NOTES:

1. Apparent conductivity in millimhos/meter.
2. Data from EM 31.
   Coil spacing 3.7M

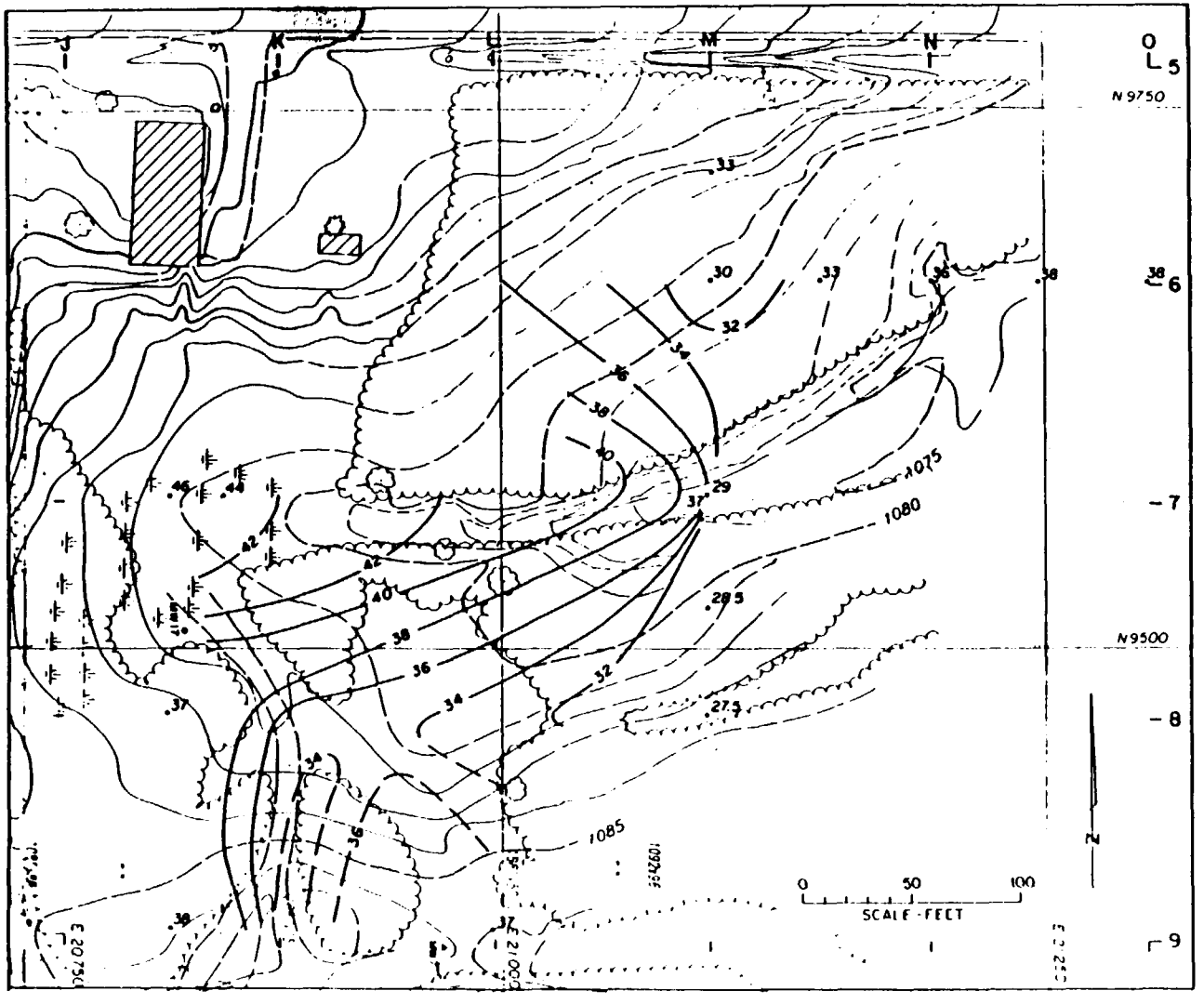
FIGURE 8
CONTOUR MAP
EM-31 (OFF-SITE)
SUMMIT NATIONAL SITE



NOTES:

1. Apparent conductivity in millimhos/meter.
2. Data from EM 34-3.
   Coil spacing 10M.
   Coils vertical.

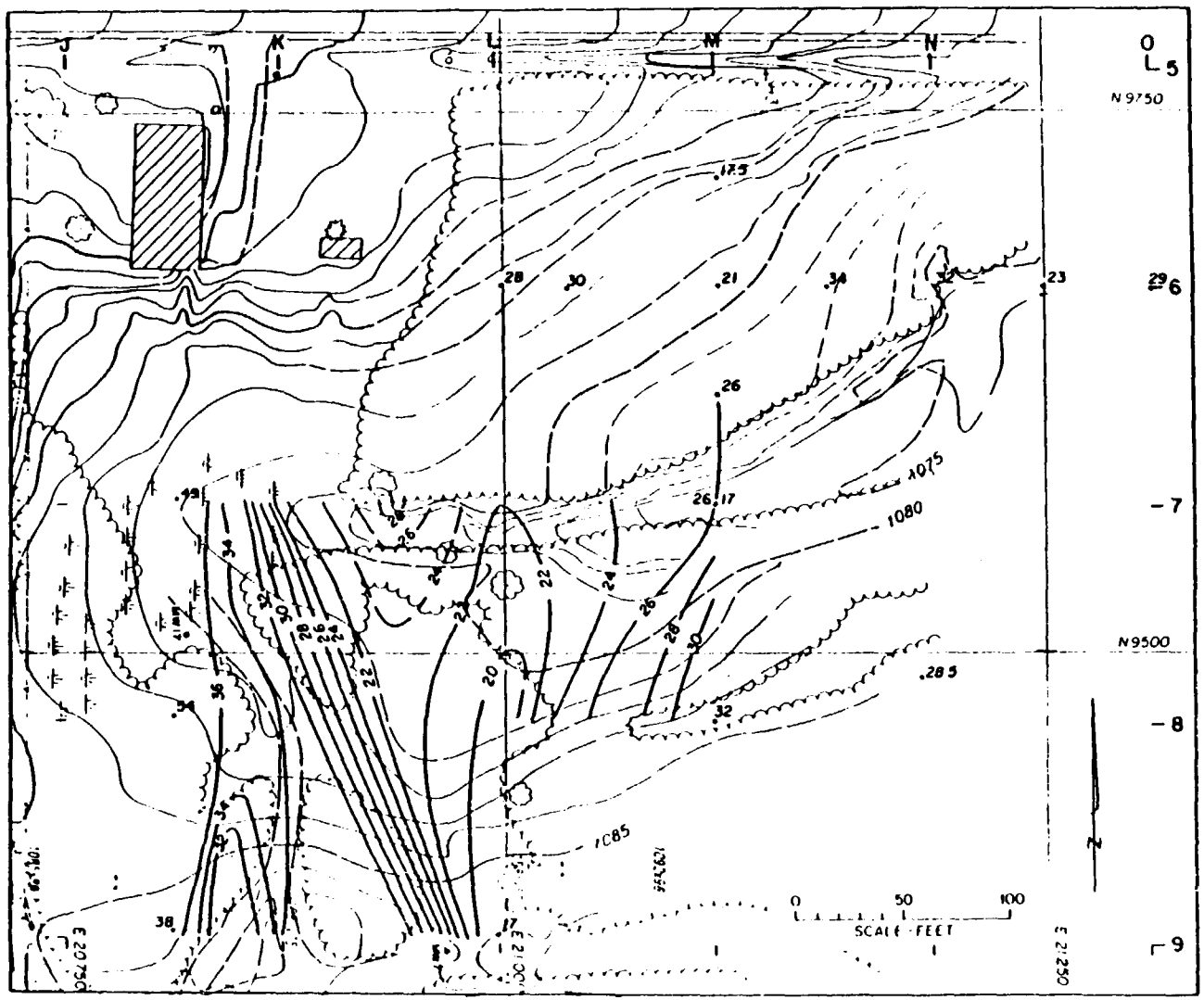
FIGURE 9
CONTOUR MAP EM 34-3 (OFF-SITE)
(10M COIL SEPERATION, VERTICALS)
SUMMIT NATIONAL SITE



NOTES:

1. Apparent conductivity in millimhos/meter.
2. Data from EM 34-3.
   Coil spacing 20M.
   Coils vertical.

FIGURE 10
CONTOUR MAP EM 34-3 (OFF-SITE)
(20M COIL SEPERATION, VERTICAL COILS)
SUMMIT NATIONAL SITE



NOTES:

1. Apparent conductivity in millimhos/meters.
2. Data from EM 34-3.
   Coil spacing 20M.
   Coils horizontal.

FIGURE 11
CONTOUR MAP EM 34-3 (OFF-SITE)
(20M COIL SEPERATION, HORIZONTAL COILS)
SUMMIT NATIONAL SITE

APPENDIX A

MAGNETOMETER [TOTAL FIELD] MEASUREMENTS FOR DETECTION OF BURIED METAL OBJECTS METHOD OF INVESTIGATION

Introduction

The magnetic method is a versatile, relatively inexpensive, geophysical exploration technique. Aeromagnetic surveys and deep water marine studies are commonly used as a reconnaissance tool for tracing large-scale geologic structure. Land and coastal water marine data are more useful in tracing smaller, more localized geologic structures, such as mineral and ore deposits. Land and marine surveys yield more detail and higher resolution, since the measurements are taken closer to the anomaly source. Land and shallow water magnetic data is commonly used to locate larger, buried, man-made objects such as pipelines, barrels or other buried metal objects, and smaller objects such as involved in archealogical prospecting.

Earth Magnetism

Magnetics is a "potential field" method. For a given magnetic field, the magnetic force in a given direction is equal to the derivative of the magnetic potential in that direction. The source of the earth's magnetic potential is its own magnetic field and the induction effect this field has on magnetic objects or bodies above and below the surface. The earth's field is a vector quantity having a unique magnitude and direction at every point on the earth's surface. This magnetic field is defined in three dimensions by angular quantities known as declination and inclination. Declination is defined as the angle between geographic north and magnetic north, and inclination is the angle between the direction of the earth's field and the horizontal [Figure 1]. The earth's magnetic field is measured in "gammas" [where 1 gamma = 10^{-5} Oersted]; the total field ranges from about 25,000 gammas near the equator to 70,000 gammas near the poles.

The earth's magnetic field is not completely stable. It undergoes long-term [secular] variations over centuries; small, daily [diurnal] variations [less than 1% of the total field magnitude]; and transient fluctuations called magnetic storms resulting from solar flare phenomena.

The earth's ambient magnetic field is modified locally by both naturally-occurring and man-made magnetic materials. Iron or steel objects act as "local" dipoles, which are generally oriented differently than the earth's external magnetic field.

The iron or steel objects represents a local perturbation in the main earth field. The net field in the vicinity of this perturbation is simply the vector sum of the induced and earth fields. Thus, the induced field is a function of the "susceptibility" of the material, or its ability to act like a magnet.

Remanent magnetization is produced in materials which have been heated above the Curie point allowing magnetic minerals in the material to become aligned with the earth's field before cooling. The remanent field direction is not always parallel to the earth's present field, and can often be completely reversed. The remanent field combines vectorially with the ambient and induced field components. The contribution of the remanent components must be considered in magnetic interpretations.

Instrumentation

At present, the most widely used magnetometer is the "proton precession" type. This device utilizes the precession of spinning protons of the hydrogen atoms in a sample of fluid [kerosene, alchohol, or water] to measure total magnetic field intensity.

Protons spinning in an atomic nucleus behave like magnetic dipoles, which are aligned [polarized] in a uniform magnetic field. The protons initially aligned themselves parallel to the earth's field. A second, much stronger magnetic field is produced approximately perpendicular to the earth's field by introducing current through a coil of wire. The protons become temporarily aligned with this stronger secondary field. When this secondary field is removed, the protons tend to realign [precess] themselves parallel to the earth's field direction. The precessing protons will generate a small electric signal in the same coil used to polarize them with a frequency [about 2,000 Hz] proportional to the total magnetic field intensity but independent of the coil

orientation. By measuring the signal frequency, the absolute value of the total earth field intensity can be obtained to a 1 gamma accuracy. The total magnetic field value measured by the proton precession magnetometer is the net vector sum of the ambient earth's field and any local induced and/or remanent perturbations.

A total field proton precession magnetometer can be made portable and does not require orientation or leveling. There are a few limitations associated with the precession system. The precession signal can be severely degraded in the presence of large field gradients [greater than 200 gammas per foot] are near 60-cycle A/C power lines. Also, the interpretation of total field data is sometimes more complicated than vertical field data which, however, is more time consuming to take.

Field Techniques

The field operator must avoid or note any sources of high magnetic gradients and alternating currents, such as power lines, buildings, and any large iron or steel objects. Readings are taken at a predetermined interval which depends on the nature of the survey, the accuracy required, and the gradients encountered. Base station readings, if required, are usually made several times a day to check for diurnal variations and magnetic storms.

Interpretation

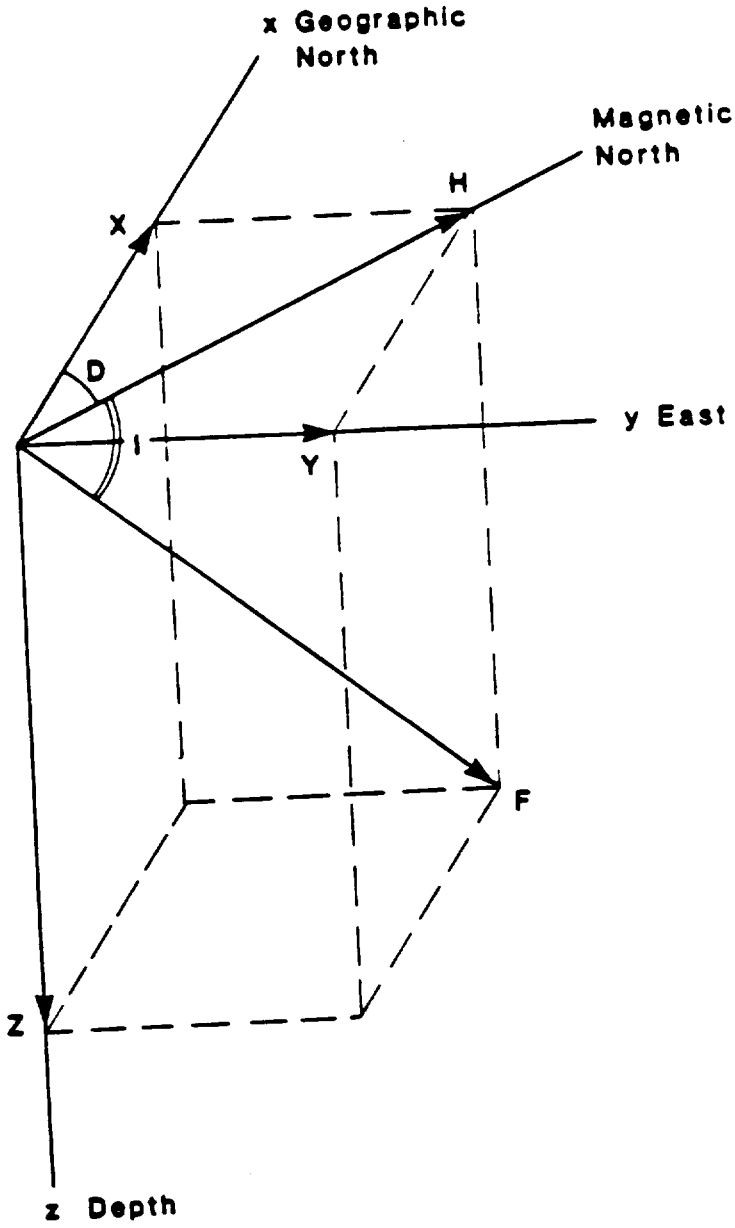
Lateral variations in susceptibility and/or remanent magnetization in crustal rocks give rise to localized anomalies in the measured total magnetic field intensity. Geologic structural features [faults, contacts, intrusions, etc.] and metal objects will cause magnetic anomalies, which can be interpreted to define the location of the causative source.

After diurnal effects and regional gradients have been removed, magnetic anomalies can be studied in detail with derivative operations and frequency filtering employed to define depth and shape.

Because it is a potential field method, there are a number of possible source configurations for any given magnetic anomaly. There is also an inherent complexity in magnetic dipole behavior. If the various magnetic field parameters [inclination, declination, and susceptibility] are well defined, and some reasonable assumptions can be made regarding the nature of the source, an accurate source model can generally be derived.

Magnetic anomalies can be analyzed both qualitatively and quantitatively. The physical dimensions of an anomaly [slope, wave-length, amplitude, etc.] often reveal enough to draw some general qualitative conclusions regarding the location and depth of the causative source.

Precise interpretation must be done quantitatively and requires prior knowledge of earth and remanent magnetic field parameters. Modeling can be performed by various approximation methods, whereby one reduces the source to a system of poles or dipoles, or assumes it to be one of several simple, geometric forms [vertical prism, horizontal slab, step, etc.]. The magnetic properties for this simplified model can be rather easily defined mathematically. Simple formulas can be derived which relate readily measurable anomaly parameters, such as slope, width, and amplitude ratios, to the general dimensions of the anomaly source, including depth to top, thickness, dip, and width normal to strike. Since these methods involve very limiting geometric assumptions, the results can be treated as good approximations only for very simplified sources.



I = Inclination

D = Declination

H = Horizontal Field Strength

F = Total Magnetic Force

ELEMENTS OF THE EARTH'S MAGNETIC FIELD

FIGURE 1

APPENDIX B

ELECTROMAGNETIC TERRAIN CONDUCTIVITY METHOD OF INVESTIGATION

GENERAL CONSIDERATIONS

The electromagnetic terrain conductivity [EM] survey is a method of obtaining subsurface information through electric measurements made at the surface of the earth. The parameter measured with this technique is the apparent conductivity of the subsurface. The conductivity meter consists of receiver coil and a separate transmitter coil which induces an electrical source field [a circular eddy current loop] in the earth [Figure A-1]. Each one of the current loops generates a magnetic field proportional to the value of the current flowing within the loop. A part of the magnetic field from each current loop is intercepted by the receiver coil and converted to an output voltage which is linearly related to terrain conductivity. The instrument is calibrated to permit direct reading of conductivity values in millimhos per meter. The conductivity meter has a variable operating frequency, from 9.8 KH_z for the EM31 to a range of 6.4 KH_z to 0.4 KH_z for the EM34-3. The measurement precision is $\pm$ 2% of full scale with a measurement accuracy of + 5% at 20 millimhos per meter. The operating frequency of the EM34-3 decreases as the coil spacing increases. Coil spacings of 10, 20 and 40 meters are standard with the EM34-3.

Geologic materials have unique electrical characteristics and lateral variations in conductivity values indicate a change in subsurface conditions. The relative conductivity of earth materials is proportional to their content of water and dissolved salts or ions. Accordingly, dry sands and gravels, and massive rock formations would have low conductivity values; conversely, most clays and materials with a high ion content would have high conductivity values.

FIELD PROCEDURE FOR DATA ACQUISITION

Conductivity measurements are generally made at 10 meter spacings along a survey line to yield good spatial resolution. Readings taken with the EM31 at a height of one meter above ground surface are sufficiently accurate but for maximum accuracy the instrument can be read at the ground surface. Readings obtained with the EM34-3 are commonly made with the coils in the vertical coplanar configuration, horizontal dipole mode [HDM]. Deeper, approximately 15 meters vs. 7.5 meters, penetration with an equivalent coil spacing can be achieved by using the horizontal coplanar configuration, vertical dipole mode [VDM].

APPENDIX C

GROUND PENETRATING RADAR [GPR] Survey
METHOD OF INVESTIGATION

Ground penetrating radar is an electromagnetic survey technique that reveals a graphic cross sectional view of layered material below the ground surface. It is an echo-ranging technique similar to the single-trace reflection method commonly used in marine subbottom profiling in which reflective layers are traced by echo patterns generated in response to acoustic impulses. The two techniques differ in that the acoustic method uses audio frequency sound waves transmitted through a water medium to the material under investigation. The radar method transmits, directly to the surface, impulses of radio waves at frequencies up to a thousand megahertz.

In a radar system [Figure 1], high-frequency impulses of radio energy are generated by the transmitter. A beam of these impulses is emitted by a special antenna placed in close proximity to the ground so that it couples electromagnetically to the surface material. Each impulse propagates downward through the ground surface and into the material below. At interfaces, part of the signal is reflected while part is transmitted still deeper to be reflected by other layers or isolated bodies. For each impulse transmitted, a string of reflected impulses is returned to the antenna in a time sequence proportional to the round-trip travel time to each reflector. After transmitting the outgoing pulse, the system instantly switches from the transmitter to the receiver in order to detect the echo signals. When operated in the field during data acquisition, a graphic recorder provides an immediate view of the data. Data enhancement is possible if the data are recorded on a magnetic tape recorder for later playback at a slower speed.

Ground penetrating radar surveys are carried out by pulling the antenna slowly along a pre-measured survey line. Radar impulses are transmitted in synchronism with a swept-stylus type graphic recorder. The graphic recorder stylus sweeps across the paper at a uniform speed and echo signals cause the paper to be darkened at points proportional to the total travel time to the reflector producing the echo. Because the antenna is being pulled forward, each pass of the stylus represents a slightly different antenna position. As the recorder paper is pulled under the moving stylus, a pattern of reflective interfaces is generated.

Figure 2 shows a typical record and a representation of the radar echo impulses. The recorder detects the presence of an echo whenever the signal level exceeds a preset threshold. The paper is darkened to coincide with every exceedance along any given sweep of the stylus. The pattern of darkened regions on the paper marks the reflective horizons in the earth. The distance shown on the recorder paper to any reflector is proportional to its depth below the path of the antenna.

Accurate determination of the depth to any layer requires cali- bration of the radar system.

The depth to an identified reflector such as a pipe, a barrel or a geologic feature, is the most direct and easiest method available for vertical scale calibration.

If the depth to an observed reflector is not known, a borehole can be drilled to establish its depth. This is a more costly procedure, but it provides an exact depth calibration at each drill location and also allows propagation velocity and a more precise dielectric constant to be determined.

Depth of penetration in a given material is limited by attenuation of the signal. Attenuation is a function of dielectric loss and of electrical conductivity loss which, in a given material, will vary with the amount of water, dissolved salt, temperature, density, and frequency of the radar impulses. Penetration of up to 75 feet has been reported for water saturated sands in a Massachusetts glacial delta. The antarctic ice shelf has been penetrated to 230 feet. Wet clays, how- ever, will attenuate the signal within five feet, and sea water is transparent to less than one foot. It is important to note that in a layered material a single, highly reflective layer alone can limit penetration by preventing the propagation of energy past it. In this case, apparent loss of energy is caused by reflection rather than by dissipation.

Radar has been used to locate underground pipes, foundations, voids, sand, gravel, peat, and archeological artifacts. Layered structures in soils and hard rock have been accurately charted in long continuous profiles. The ease with which modern systems can be used makes ground penetrating radar a logical choice where rapid and accurate shallow surveys are required.

TABLE 1

Table of Typical Electromagnetic Constants
for Earth Materials at Radio Frequencies

Material	Dielectric Constant [relative]
Air	1
Clay	8-12
Fresh Water	80
Sea Water	80
Granite, dry	5-19
Granite, typical	15
Ice	4
Limestone	7-15
Sand, Dry	3
Sandstone, 4% water	11
Sandstone, dry	5
Soil, 42% water	30
Soil, 8% water	4

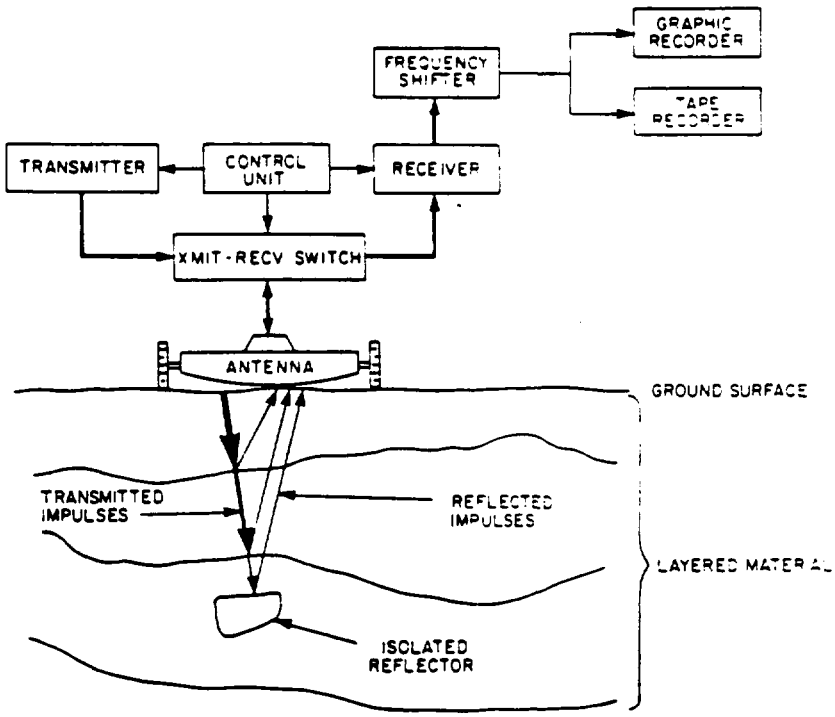


FIGURE 1. RADAR SYSTEM BLOCK DIAGRAM

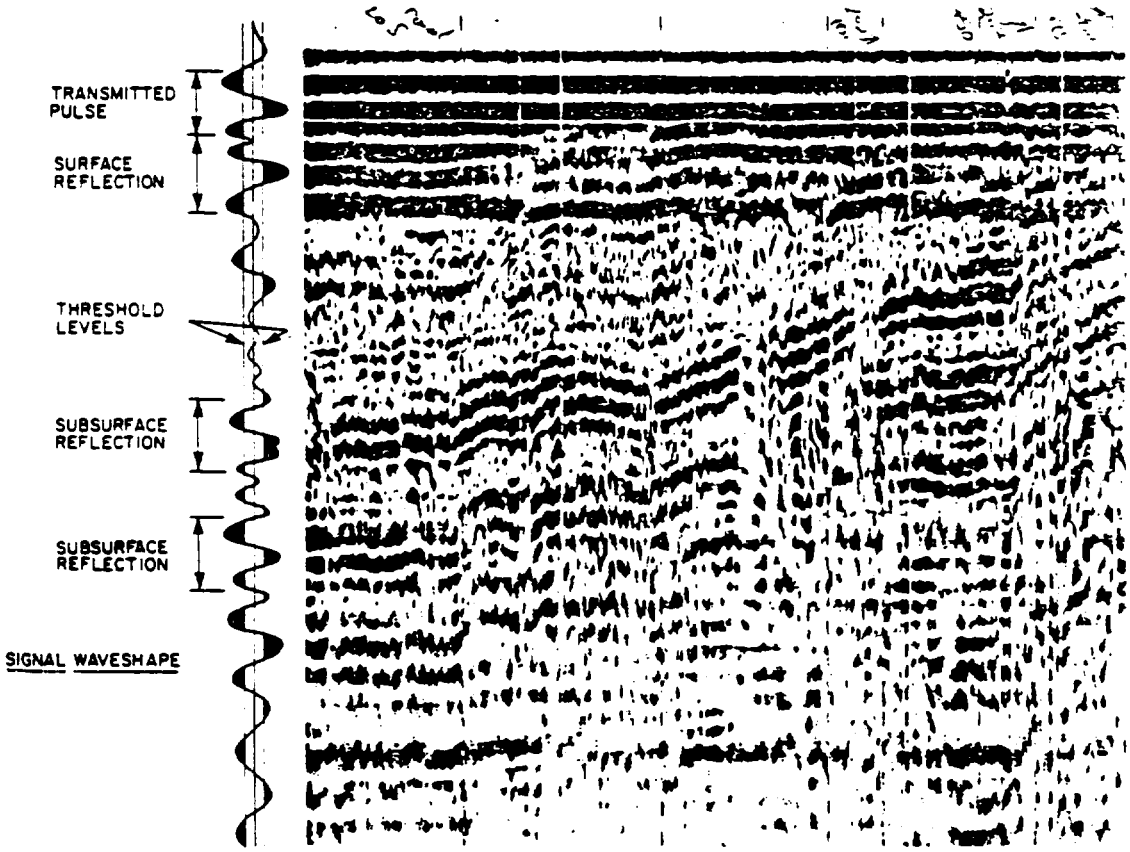


FIGURE 2. TYPICAL RADAR RECORD

Appendix H

APPENDIX H - PUBLIC HEALTH EVALUATION APPENDICES

PHE1	Summaries of Contaminants Found On Site
PHE2	Chemical Toxicity Profiles
PHE3	Estimated Soil Ingestion Rates for Use in Risk Assessments

APPENDIX PHE 1

Table 1 - Ranking of Selected Chemicals Detected in Surface Soil

	Frequency of Detection (b)	Concentration Minimum (ug/kg)	Concentration Maximum (ug/kg)	Toxicity Constant (c) (kg/ug)	Indicator Score (d)
Ranking Based on Potential Carcinogenic Effects					
PCBs	14/47	40 -	590000	2.86E-08	1.69E-02
arsenic*	40/47	7300 -	35000	2.03E-07	7.11E-03
hexachlorobenzene	11/47	280 -	32000	1.68E-08	5.38E-04
polycyclic aromatic hydrocarbons (PAHs)	13/47	150 -	1600	2.28E-07	3.65E-04
1,2-dichloroethane	6/47	44 -	23000	2.93E-09	6.74E-05
bis (2-ethylhexyl) phthalate	34/47	550 -	1400000	2.86E-11	4.00E-05
trichloroethene	28/47	2 -	86000	2.14E-10	1.84E-05
tetrachloroethene	11/47	1 -	4600	4.43E-10	2.04E-06
chloroform	6/47	2 -	42	2.81E-09	1.18E-07

Ranking Based on Noncarcinogenic Effects	Frequency	Minimum	Maximum	Toxicity Constant	Indicator Score
bis (2-ethylhexyl) phthalate	34/47	550 -	1400000	2.23E-07	3.12E-01
antimony	10/47	16000 -	545000	2.17E-07	1.18E-01
barium	47/47	47000 -	244000	2.04E-07	4.98E-02
arsenic	40/47	7300 -	35000	9.00E-07	3.15E-02
cadmium	7/47	3000 -	112000	2.23E-07	2.50E-02
nickel	44/47	11000 -	56000	2.13E-07	1.19E-02
lead	47/47	10000 -	116000	4.46E-08	5.17E-03
trichloroethene	28/47	2 -	86000	5.26E-08	4.52E-03
zinc	47/47	26000 -	903000	5.33E-09	4.28E-03
copper	47/47	10000 -	67000	3.57E-08	2.39E-03
PAHs	13/47	150 -	1600	1.33E-06	2.13E-03
mercury	28/47	100 -	800	9.21E-07	7.37E-04
hexachlorobenzene	11/47	280 -	32000	2.00E-08	6.40E-04
vanadium	47/47	14000 -	62000	7.14E-09	4.43E-04
phenol	5/47	290 -	44000	5.02E-09	2.21E-04
toluene	29/47	3 -	260000	2.60E-10	6.76E-05
chlorobenzene	7/47	4.9 -	3600	7.14E-09	2.57E-05
ethyl benzene	13/47	3 -	36000	5.52E-10	1.99E-05
tetrachloroethene	11/47	1 -	4600	4.81E-10	2.21E-06
benzene	27/47	1 -	21	5.85E-09	1.23E-07
dichloromethane	38/47	2 -	910	4.60E-11	4.19E-08
1,2-dichloroethane	6/47	2 -	42	8.80E-10	3.70E-08
diethyl phthalate	8/47	330 -	1600	1.34E-11	2.14E-08
cyanide	30/47	310 -	43600		
acetone	21/47	6 -	8200		
beryllium	23/47	640 -	1900		
iron	47/47	11489000 -	95300000		
total xylenes	18/47	7.8 -	210000		
hexachlorocyclopentadiene	1/47		53000		
manganese	47/47	29000 -	2620000		
isophorone	3/47	800 -	3000		
chromium	47/47	10000 -	102000		

(a) 1984 surface soil data
(b) Frequency = # detected/#sampled
(c) USEPA (1986)
(d) Indicator score = maximum concentration X toxicity constant

Table 2 - Ranking of Selected Chemicals Detected in Subsurface Soil [a]

	Frequency of Detection (b)	Concentration Minimum (ug/kg)	Concentration Maximum (ug/kg)	Toxicity Constant (c) (kg/ug)	Indicator Score (d)
Ranking Based on Potential Carcinogenic Effects					
PCBs (arochlor 1248)	5/26	990 -	310000	2.86E-08	8.87E-03
arsenic	26/26	12000 -	27000	2.03E-07	5.48E-03
hexachlorobenzene	7/26	96 -	59000	1.68E-08	9.91E-04
polycyclic aromatic hydrocarbons (PAHs)	16/26	43 -	630	2.29E-07	1.44E-04
1,2-dichloroethane	8/26	21 -	8900	2.93E-09	2.61E-05
trichloroethene	19/23	5 -	99000	2.14E-10	2.12E-05
bis (2-ethylhexyl) phthalate	25/26	47 -	330000	2.86E-11	9.44E-06
1,1-dichloroethene	3/26	3 -	430	6.14E-09	2.64E-06
tetrachloroethene	5/26	3 -	2500	4.43E-10	1.11E-06

Ranking Based on Noncarcinogenic Effects	Frequency	Minimum	Maximum	Toxicity Constant	Indicator Score
bis (2-ethylhexyl) phthalate	25/26	47 -	330000	2.23E-07	7.36E-02
barium	26/26	35000 -	245000	2.04E-07	5.00E-02
arsenic*	26/26	12000 -	27000	9.00E-07	2.43E-02
nickel	26/26	10000 -	42000	2.13E-07	8.95E-03
trichloroethene	19/26	5 -	99000	5.26E-08	5.21E-03
silver	7/26	2600 -	4200	1.00E-06	4.20E-03
lead	26/26	12000 -	84000	4.46E-08	3.75E-03
cadmium	9/26	2400 -	13000	2.23E-07	2.90E-03
zinc	26/26	34000 -	448000	5.33E-09	2.39E-03
copper	26/26	22000 -	51000	3.57E-08	1.82E-03
hexachlorobenzene	7/26	96 -	59000	2.00E-08	1.18E-03
polycyclic aromatic hydrocarbons (PAHs)	16/26	43 -	630	1.33E-06	8.38E-04
mercury	6/26	84 -	810	9.21E-07	7.46E-04
vanadium	26/26	11000 -	26000	7.14E-09	1.86E-04
1,1-dichloroethane	8/26	5 -	41000	1.29E-09	5.29E-05
ethyl benzene	20/26	4 -	76000	5.52E-10	4.20E-05
phenol	6/26	52 -	7800	5.02E-09	3.92E-05
chlorobenzene	4/26	3 -	5200	7.14E-09	3.71E-05
toluene	26/26	17 -	140000	2.60E-10	3.64E-05
1,2,4-trichlorobenzene	4/26	100 -	1400	1.07E-08	1.50E-05
1,1-dichloroethene	3/26	3 -	430	1.86E-08	8.00E-06
1,2-dichloroethane	8/26	29 -	8900	8.80E-10	7.83E-06
tetrachloroethene	5/26	3 -	2500	4.81E-10	1.20E-06
1,1,1-trichloroethane	14/26	6 -	24000	3.67E-11	8.81E-07
dichloromethane	26/26	5 -	5100	4.60E-11	2.35E-07
carbon disulfide	9/26	3 -	10	2.12E-08	2.12E-07
cyanide	5/26	1100 -	8800		
beryllium	23/26	490 -	1600		
acetone	25/26	26 -	48000		
manganese	26/26	57000 -	683000		
total xylenes	26/26	9 -	270000		
iron	26/26	16100000 -	50800000		
chromium	26/26	9000 -	732000		

(a) 1986 subsurface soil data
(b) Frequency = # detected/#sampled
(c) USEPA (1986)
(d) Indicator score = maximum concentration X toxicity score

Table 3 - Ranking of Selected Chemicals Detected in Groundwater [a]

Ranking Based on Potential Carcinogenic Effects	Frequency of Detection (b)	Concentration Minimum (ug/l)	Concentration Maximum (ug/l)	Toxicity Constant (c) (l/ug)	Indicator Score (d)
1,2-dichloroethane	10/23	2 -	130000	5.86E-05	7.62E+00
polycyclic aromatic hydrocarbons (PAHs)	1/23		77	4.55E-03	3.50E-01
1,1-dichloroethylene	3/23	1 -	2600	1.23E-04	3.20E-01
trichloroethylene	5/23	4 -	27000	4.29E-06	1.16E-01
arsenic (e)	4/23	12 -	19	4.07E-03	7.73E-02
bis (2-ethylhexyl) phthalate	19/23	3 -	9600	5.71E-07	4.91E-03

Ranking Based on Noncarcinogenic Effects	Frequency	Minimum	Maximum	Toxicity Constant	Indicator Score
trichloroethylene	5/23	4 -	27000	1.00E-03	2.70E+01
barium	13/23	4.8 -	1550	4.08E-03	6.32E+00
nickel	11/23	12 -	1120	4.26E-03	4.77E+00
1,2-dichloroethane	10/23	2 -	130000	1.76E-05	2.29E+00
polycyclic aromatic hydrocarbons (PAHs)	1/23		77	2.67E-02	2.06E+00
1,1-dichloroethylene	3/23	1 -	2600	3.71E-04	9.65E-01
phenol	5/23	260 -	7000	1.00E-04	7.00E-01
zinc	22/23	5.5 -	3750	1.07E-04	4.01E-01
arsenic	4/23	12 -	19	1.80E-02	3.42E-01
1,1-dichloroethane	9/23	2 -	12000	2.58E-05	3.10E-01
ethyl benzene	4/23	1 -	11000	1.10E-05	1.21E-01
toluene	13/23	1 -	20000	5.20E-06	1.04E-01
1,1,1-trichloroethane	7/23	3 -	53000	7.33E-07	3.88E-02
dichloromethane	23/23	1 -	24000	9.20E-07	2.21E-02
copper	2/23	6.1 -	20	7.14E-04	1.43E-02
vanadium	4/23	7.1 -	47	1.43E-04	6.72E-03
di-n-butyl phthalate	9/23	2 -	130	3.81E-05	4.95E-03
isophorone	7/23	3 -	2600		
chromium	7/23	4.2 -	55		
iron	20/23	24 -	982000		
acetone	23/23	2 -	1300000		
manganese	19/23	14 -	72200		
bis (2-ethylhexyl) phthalate	19/23	3 -	9600		

(a) 1986 groundwater data
(b) Frequency = #detected/#sampled
(c) USEPA (1986)
(d) Indicator score = maximum concentration X toxicity constant
(e) All metals concentrations are dissolved concentrations

Table 4 - Ranking of Selected Chemicals Detected in Groundwater [a]

Ranking Based on Potential Carcinogic Effects	Frequency of Detection (b)	Concentration Minimum (ug/l)	Concentration Maximum (ug/l)	Toxicity Constant (c) (l/ug)	Indicator Score (d)
1,2-dichloroethane	10/23	2 -	130000	5.86E-05	7.62E+00
polycylcic aromatic hydrocarbons (PAHs)	1/23		77	4.55E-03	3.50E-01
1,1-dichloroethylene	3/23	1 -	2600	1.23E-04	3.20E-01
arsenic (e)	9/23	13 -	72	4.07E-03	2.93E-01
trichloroethylene	5/23	4 -	27000	4.29E-06	1.16E-01
bis (2-ethylhexyl) phthalate	19/23	3 -	8600	5.71E-07	4.91E-03

Ranking Based on Noncarcinogenic Effects	Frequency	Minimum	Maximum	Toxicity Constant	Indicator Score
trichloroethylene	5/23	4 -	27000	1.00E-03	2.70E+01
barium	20/23	12 -	3520	4.08E-03	1.44E+01
nickel	20/23	7.5 -	1210	4.26E-03	5.15E+00
1,2-dichloroethane	10/23	2 -	130000	1.76E-05	2.29E+00
PAHs	1/23		77	2.67E-02	2.06E+00
arsenic	9/23	13 -	72	1.80E-02	1.30E+00
1,1-dichloroethylene	3/23	1 -	2600	3.71E-04	9.65E-01
phenol	5/23	260 -	7000	1.00E-04	7.00E-01
zinc	23/23	24 -	4360	1.07E-04	4.67E-01
copper	21/23	6.8 -	502	7.14E-04	3.58E-01
1,1-dichloroethane	9/23	2 -	12000	2.58E-05	3.10E-01
lead	5/23	15 -	220	8.93E-04	1.96E-01
ethyl benzene	4/23	1 -	11000	1.10E-05	1.21E-01
toluene	13/23	1 -	20000	5.20E-06	1.04E-01
vanadium	15/23	5.2 -	321	1.43E-04	4.59E-02
1,1,1-trichloroethane	7/23	3 -	53000	7.33E-07	3.88E-02
dichloromethane	23/23	1 -	24000	9.20E-07	2.21E-02
mercury	9/23	0.3 -	0.9	1.84E-02	1.66E-02
di-n-butyl phthalate	9/23	2 -	130	3.81E-05	4.95E-03
chromium	18/23	4.1 -	318		
bis (2-ethylhexyl) phthalate	19/23	3 -	8600		
manganese	22/23	21 -	69500		
beryllium	6/23	2.5 -	22		
iron	23/23	160 -	1020000		
isophorone	7/23	3 -	2600		
acetone	23/23	2 -	1300000		
cyanide	4/23	16 -	239		

(a) 1986 groundwater data
(b) Frequency = # detected/#sampled
(c) USEPA (1986)
(d) Indicator score = maximum concentration X toxicity constant
(e) All metals concentrations are total concentrations

Table 5 - Ranking of Selected Chemicals Detected in Sediment

Ranking Based on Potential Carcinogenic Effects	Frequency of Detection (b)	Concentration Minimum (ug/kg)	Concentration Maximum (ug/kg)	Toxicity Constant (c) (kg/ug)	Indicator Score (d)
arsenic	10/12	14000 -	38000	2.03E-07	7.71E-03
PCBs	3/12	4063 -	21000	2.86E-08	6.01E-04
polycyclic aromatic hydrocarbons (PAHs)	1/12		309	2.28E-07	7.05E-05
1,2-dichloroethane	7/12	3 -	16608	2.93E-09	4.87E-05
hexachlorobenzene	1/12		518	1.68E-08	8.70E-06
bis (2-ethylhexyl) phthalate	11/12	997 -	291808	2.86E-11	8.35E-06
heptachlor epoxide	1/12		8.1	4.14E-07	3.35E-06
1,1-dichloroethene	4/12	13 -	15	6.14E-09	9.21E-08
vinyl chloride	1/12		102	2.14E-10	2.18E-08
benzene	2/12	10 -	25	3.86E-10	9.65E-09
trichloroethene	3/12	10 -	20	2.14E-10	4.28E-09

Ranking Based on Noncarcinogenic Effects	Frequency	Minimum	Maximum	Toxicity Constant	Indicator Score
bis (2-ethylhexyl) phthalate	11/12	997 -	291808	2.23E-07	6.51E-02
barium	12/12	31000 -	170000	2.04E-07	3.47E-02
arsenic	10/12	14000 -	38000	9.00E-07	3.42E-02
antimony	9/12	52000 -	143000	2.17E-07	3.10E-02
nickel	12/12	15000 -	65000	2.13E-07	1.38E-02
vinyl chloride	1/12		102	8.77E-05	8.95E-03
zinc	12/12	82000 -	1570000	5.33E-09	8.37E-03
lead	12/12	23000 -	92000	4.46E-08	4.10E-03
copper	12/12	34000 -	74000	3.57E-08	2.64E-03
PAHs	1/12		309	1.33E-06	4.11E-04
vanadium	11/12	13000 -	31000	7.14E-09	2.21E-04
di-n-butyl phthalate	7/12	339 -	55738	1.90E-09	1.06E-04
1,2-dichloroethane	7/12	3 -	16608	8.80E-10	1.46E-05
hexachlorobenzene	1/12		518	2.00E-08	1.04E-05
1,1-dichloroethane	3/12	69 -	2261	1.29E-09	2.92E-06
chlorobenzene	4/12	8 -	329	7.14E-09	2.35E-06
trichloroethene	3/12	10 -	20	5.26E-08	1.05E-06
1,1-dichloroethene	4/12	13 -	15	1.86E-08	2.79E-07
benzene	2/12	10 -	25	5.85E-09	1.46E-07
ethyl benzene	2/12	20 -	74	5.52E-10	4.08E-08
dichloromethane	12/12	8 -	870	4.60E-11	4.00E-09
1,1,1-trichloroethane	9/12	27 -	787	3.67E-11	2.89E-08
toluene	2/12	20 -	74	2.60E-10	1.92E-08
chromium	12/12	10000 -	73000		
cyanide	7/12	2400 -	74000		
iron	12/12	31800000 -	166000000		
acetone	8/12	9 -	240		
manganese	12/12	112000 -	2810000		
total xylenes	3/12	4 -	67		

(a) 1985-6 sediment data
(b) Frequency = #detected/#sampled
(c) USEPA (1986)
(d) Indicator score = maximum concentration X toxicity constant

Table 6 - Ranking of Selected Chemicals Detected in Surface Water [a]

Ranking Based on Potential Carcinogenic Effects	Frequency of Detection (b)	Concentration Minimum (ug/l)	Concentration Maximum (ug/l)	Toxicity Constant (c) (l/ug)	Indicator Score (d)
arsenic (e)	1/6		25	4.07E-03	1.02E-01
polycyclic aromatic hydrocarbons (PAHs)	1/6		3	4.55E-03	1.37E-02
1,2-dichloroethane	4/6	38 -	78	5.86E-05	4.57E-03
tetrachloroethene	1/6		24	8.86E-06	2.13E-04
hexachloroethane	1/6		14	2.29E-06	3.21E-05
vinyl chloride	1/6		7	4.29E-06	3.00E-05
trichloroethene	1/6		6	4.29E-06	2.57E-05
bis (2-ethylhexyl) phthalate	6/6	4 -	14	5.71E-07	7.99E-06

Ranking Based on Noncarcinogenic Effects	Frequency	Minimum	Maximum	Toxicity Constant	Indicator Score
selenium	1/6		16	1.05E-01	1.68E+00
nickel	5/6	20 -	322	4.26E-03	1.37E+00
arsenic	1/6		27	1.80E-02	4.86E-01
antimony	1/6		43	4.35E-03	1.87E-01
zinc	6/6	15 -	1660	1.07E-04	1.78E-01
barium	5/6	9.9 -	34	4.08E-03	1.39E-01
cadmium	1/6		23	4.45E-03	1.02E-01
copper	6/6	9.4 -	122	7.14E-04	8.71E-02
PAHs	1/6		3	2.67E-02	8.01E-02
chlorobenzene	2/6	2 -	59	1.43E-04	8.44E-03
trichloroethene	1/6		6	1.00E-03	6.00E-03
1,2-dichloroethane	4/6	38 -	78	1.76E-05	1.37E-03
vanadium	1/6		8.3	1.43E-04	1.19E-03
1,1-dichloroethane	3/6	3 -	34	2.58E-05	8.77E-04
vinyl chloride	1/6		7	8.77E-05	6.14E-04
tetrachloroethene	1/6		24	9.62E-06	2.31E-04
hexachloroethane	1/6		14	6.62E-06	9.27E-05
1,1,1-trichloroethane	3/6	5 -	29	7.33E-07	2.13E-05
toluene	3/6	1 -	2	5.20E-06	1.04E-05
beryllium	1/6		7.9		
chromium	3/6	4.2 -	22		
total xylenes	2/6		1		
bis (2-ethylhexyl) phthalate	6/6	4 -	14		
isophorone	1/6		4		
manganese	6/6	212 -	7980		
iron	6/6	657 -	131000		
acetone	6/6	15 -	47		

(a) 1986 onsite and downstream surface water data
(b) Frequency = # detected/# sampled
(c) USEPA (1986)
(d) Indicator score = maximum concentration X toxicity constant
(e) Metals concentrations are dissolved concentrations

APPENDIX PHE 2

ACETONE

INTRODUCTION

Acetone is a widely used industrial solvent that is found in paints, varnishes, and lacquers. The rapid evaporation rate of acetone makes it useful for cleaning and drying precision parts.

PHARMACOKINETICS

Several studies in humans and animals indicate that acetone is absorbed through the oral and pulmonary routes of exposure (EPA 1984). Orally administered acetone is eliminated through the expired air and in the urine (EPA 1984).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Acetone has not been tested in a carcinogenicity bioassay but was found to be nonmutagenic in microbial assay systems and cell transformation systems (EPA 1984). Acetone gave negative results in assays that tested for chromosomal aberrations, sister-chromatid exchange, DNA cell binding, and point mutation in mouse lymphoma cells (EPA 1984).

No changes in clinical chemistry variables or histological changes in the liver, brain, kidneys, lungs, or heart were found in rats exposed to acetone vapors at levels of either 0 or 19,0000 ppm for 3 hours/day, 5 days/week for 8 weeks. Slight decreases in organ weights and body weights were observed in rats killed during exposure but not in rats killed 2 weeks after exposure (Bruckner and Peterson 1981).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Based on EPA guidelines for the assessment of carcinogen risk (EPA 1986), acetone is most appropriately classified Group D—Not Classified. This

category applies to agents for which there is inadequate evidence of carcinogenicity from human and animal studies.

EPA (1984) determined an acceptable intake subchronic (AIS) of 2,096 mg/day for acetone based on the inhalation study in rats by Bruckner and Peterson (1981) using a no-observed-adverse-effect level (NOAEL) OF 19,000 PPM.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) recommended a time-weighted average threshold limit value of 750 ppm (1,780 mg/m^3) for occupational exposure to acetone. The National Institute of Occupational Safety and Health (NIOSH 1978) recommended an exposure limit of 250 ppm for acetone.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

BRUCKNER, J.V., and PETERSON, R.G. 1981. Evaluation of toluene and acetone inhalant abuse. II. Modal development and toxicology. Toxicol. Appl. Pharmacol. (In Press) (As cited in EPA 1984)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Acetone. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. ECAO-CIN-H016

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Guidelines for carcinogen risk assessment. Fed. Reg. 51:33992-34003 (September 24, 1986)

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978. Criteria for a Recommended Standard—Occupational Exposure to Ketones. Washington, D.C. DHEW (NIOSH) Publication No. 78-173

ANTIMONY

INTRODUCTION

Antimony (Sb) is a brittle, silver-white metal with specific gravity 6.68, atomic number 51, and an atomic weight of 121.75 g/mole. In compounds, antimony exists in the +3 and +5 oxidation states; the most important Sb compound in commerce is Sb_2O_3, but tri-and penta-sulfides and -chlorides are also commonly utilized. Antimony compounds are utilized in therapeutics for schistosomiasis and other parasitic infections.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

The incidence of lung cancer deaths in smelter workers at the Newcastle-upon-Tyne (Great Britain) antimony and zircon works was slightly higher than expected (NIOSH 1978). However, definitive conclusions on the carcinogenicity of antimony could not be developed because the sample size was small, workers may have been exposed to other agents, and only two workers were nonsmokers. Presently available experimental studies with animals do not implicate antimony as a carcinogen.

A single report (Balyeava 1967) indicated an increase in late occurring spontaneous abortions (12.5% vs. 4.1% expected), premature births (3.4% vs. 1.2% expected), and gynecological problems (77.5% vs. 56% expected) in 318 female workers exposed to mixed dusts of antimony metal, Sb_2O_3 and Sb_2S_5, when compared with a control population. Studies reported by the same author, in which rats were administered 50 mg/kg antimony intraperitoneally or 250 mg/m^3 Sb_2O_3 dust 4 hr/day up to day 16 of gestation reported reduced litter sizes and abnormalities of the uterus and ovaries which were believed to complicate ovulation and implantation (Balyeava 1967). Casals (1972) observed no fetal abnormalities when 125 or 250 mg/kg antimony dextran glycoside was administered to pregant rats, although other reports indicate reduced rate of growth when rats were fed a diet containing 2% Sb_2O_3 (Gross et al. 1955), and a shortened life span of

rats provided 5 ppm potassium antimony tartrate in drinking water (Schroeder et al. 1970).

Cardiovascular changes associated with exposure to antimony represent a serious health effect. Exposure to either trivalent or pentavalent antimonial compounds can produce electrocardiogram (ECG) changes in humans. Histopathological evidence of cardiac edema, myocardial fibrosis, and other signs of myocardial structural damage indicates that antimony may produce even more severe, possibly permanent, myocardial damage in humans. Parallel findings of functional changes in ECG patterns and of histopathological evidence of myocardial structural damage have also been obtained in animal toxicity studies. Pneumoconiosis in response to inhalation exposure and dermatitis in response to skin exposure have also been observed among individuals exposed to antimony or its compounds.

QUANTITATIVE DESECRIPTION OF HEALTH EFFECTS

EPA (1986) calculated a reference dose (RfD) for oral exposure to antimony of 0.4 ug/kg/day (28 ug/day for the average person weighing 70 kg) based on a chronic oral bioassay in rats conducted by Schroeder et al. (1970). In this study, rats administered 0.35 mg/kg/day antimony in their drinking water exhibited decreased longevity and altered blood glucose and cholesterol levels. EPA used this study and an uncertainty factor of 1,000 to arrive at the RfD of 0.4 ug/kg/day.

NIOSH recommends a standard of 0.5 mg/m^3 in its Criteria Document (NIOSH 1978) for protection of workers from ECG alterations, dermatitis, mucous membrane irritation, and possible pneumoconiosis. The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a time-weighted average Threshold Limit Value (TLV) of 0.5 mg/m^3. However, there is uncertainty associated with this standard because of a report of increased lung cancer among workers in one smelter exposed to Sb (Davies 1973) and also reported spontaneous late abortions and premature deliveries among

Soviet women workers in antimony smelting and processing (Balyeava 1967 as cited in Stokinger 1981).

Only occupational guidelines for exposure to airborne antimony were available. Therefore, a reference concentration for antimony was calculated using the procedures outlined by EPA in their health effects assessment documents. The TLV of 0.5 mg/m^3 is multiplied by 10 m^3/20 m^3 to adjust for the difference in breathing rates between workers (10 m^3 for an 8-hour workday) and the average person (20 m^3 for 24 hours exposure) and by 5/7 to adjust for a 5 day workweek. This adjusted TLV is then divided by an uncertainty factor of 100 to protect individuals in the general population who may be especially sensitive to the chemical agent and to compensate for differences in exposure duration (workers are exposed for less than their full lifetime; the average person is assumed to be exposed for their full lifetime). Based on this calculation, the reference concentration for airborne antimony is 1.8 ug/m^3.

EPA (1980) has established an Ambient Water Criterion (AWQC) of 146 ug/liter for the protection of human health from the toxic effects of antimony ingested through water and aquatic organisms and a criterion of 45 mg/liter for antimony exposure through the ingestion of aquatic organisms only.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

BALYAEVA, A.P. 1967. The effects of antimony on reproduction. Gig. Truda Prof. Zabol. 11:32. (As cited in NIOSH 1978)

CASALS, J.B. 1972. Pharmacokinetic and toxicological studies of antimony dextran glycoside (RL-712). Brit. J. Pharmacol. 46:281. (As cited in EPA 1980)

DAVIES, T.A.L. 1973. The health of workers engaged in antimony oxide manufacture—A statement. Dept. Employment, Employment Med. Adv. Serv. (London). (As cited in EPA 1980)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Antimony. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-020

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Second Set of Verified Reference Doses. Memo from the Office of Health and Environmental Assessment to the RfD Work Group. May 14, 1986

GROSS, P., et al. 1955. Toxicological study of calcium halophosphate phosphors and antimony trioxide. I. Acute and chronic toxicity and some pharmacological aspects. Arch. Ind. Health. 11:473. (As cited in EPA 1980)

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978. Occupational Exposure to Antimony. U.S. Dept. of Health, Education, and Welfare. Publ. No. 78-216

SCHROEDER, H.A., MITCHNER, M., and NASAR, A.P. 1970. Zirconium, niobium, antimony, vanadium, and lead in rats: Lifetime studies. J. Nutr. 100:59-66. (As cited in EPA 1986)

STOKINGER, H.E. 1981. The Metals. In Clayton, G.D., and Clayton, F.E., eds. Patty's Industrial Hygiene and Toxicology. 3rd revised ed. John Wiley, New York. Pp. 1493-2060

BARIUM

INTRODUCTION

Barium is a reactive metal which exists in nature as a number of salts such as barium sulfate ($BaSO_4$) and barium carbonate ($BaCO_3$) (EPA 1984). These mineral forms are relatively insoluble in water (EPA 1985a).

PHARMACOKINETICS

In experimental animals, barium absorption varies with the species of animal, the compound tested, the age of the animal, and the dietary composition (EPA 1985a). Barium was found to distribute widely in the mouse but was principally found in the bone (Dencker et al. 1976). In humans, barium is primarily excreted via the feces (Tipton et al. 1966).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Barium carbonate and more soluble barium compounds are quite toxic to humans. Doses of barium carbonate and barium chloride of 57 mg/kg and 12 mg/kg, respectively, were fatal when ingested by humans. Toxic effects of ingestion include gastroenteritis, muscular paralysis, hypertension, cardiotoxicity including ventricular fibrillation, and damage to the central nervous system (Perry et al. 1983). Inhalation of barium sulfate or barium carbonate dust causes baritosis, a benign pneumoconiosis, in occupationally exposed workers. This effect is reversible upon cessation of exposure.

No information on the carcinogenicity, teratogenicity, mutagenicity, or chronic toxicity of barium compounds was found in the literature reviewed.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Using the EPA Carcinogen Assessment Group's criteria for evaluating weight of evidence of carcinogenicity in humans (EPA 1986), barium is most appropriately

classified in Group D--Not Classified. This category applies to agents with inadequate animal evidence of carcinogenicity.

EPA (1976) has established a maximum contaminant level (MCL) drinking water standard of 1 mg/liter for barium.

EPA (1985b) recently calculated an acceptable daily intake (ADI) for barium based on a study by Perry et al (1983) in which rats were exposed to 1, 10, or 100 mg/liter barium in drinking water for 1 to 16 months. A lowest-observed-adverse-effect level of 100 mg/liter (5.1 mg/kg/day) was identified based on the occurrence of hypertensinogenic and cardiotoxic effects. Applying an uncertainty factor of 100 (10 for interspecies extrapolation and 10 for sensitive individuals), an ADI of 0.051 mg/kg/day was recommended. An uncertainy factor of 100 rather than the traditional 1,000-fold factor was used because of the minimal exposure of the rats to trace elements (e.g., calcium) in the experiment. EPA (1985b) determined that this lack of calcium could contribute to the hypertensinogenic effects observed. Based on the ADI of 0.051 mg/kg/day and factoring in data on human exposure (0.7 mg/day via diet and 0 mg/day via air), EPA (1985b) recommended a maximum contaminant level goal (MCLG) of 1.5 mg/liter.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) has recommended a time-weighted average threshold limit value of 0.5 mg/m^3 for soluble compounds of barium and 10 mg/m^3 for barium sulfate as a nuisance dust.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

DENCKER, L., NILSSON, A., RONNBACK, C., and WALINDER, G. 1976. Uptake and retention of ^{133}Ba and ^{140}Ba-^{140}La in mouse tissue. Acta Radiol. 15:273-287 (As cited in EPA 1985a)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1976. National Interim Primary Drinking Water Regulations. Office of Drinking Water, Washington, D.C. EPA 570/9-76-003 (As cited in EPA 1985a)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Barium. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-021

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Health Advisory for Barium. Office of Drinking Water, Washington, D.C. September 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulations, synthetic organic chemicals, inorganic chemicals and microorganisms; proposed rule. Fed. Reg. 50:46936-47002 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Guidelines for carcinogen risk assessment. Fed. Reg. 51:33992-34003 (September 24, 1986)

PERRY, H.M., KOPP, S.J., ERLANGER, M.W., and PERRY, E.F. 1983. Cardiovascular effects of chronic barium ingestion. In Hemphill, D.D., ed. Trace substances in Environmental Health - XVII. Proceedings of the University of Missouri's 17th Annual Conference on Trace Substances in Environmental Health. University of Missouri Press, Columbia, Missouri (As cited in EPA 1984, 1985a)

TIPTON, R.H., STEWART, P.L., and MARTIN, P.G. 1966. Trace elements in diets and excreta. Health Phys. 12:1683-1689 (As cited in EPA 1985a)

BIS(2-ETHYLHEXYL)PHTHALATE

INTRODUCTION

Bis(2-ethylhexyl)phthalate (DEHP) is widely used as a plasticizer, primarily in the production of polyvinyl chloride resins (EPA 1980). Polyvinyl chloride resins are used in construction materials, home furnishings, transportation, apparel, and food and medical packaging materials.

PHARMACOKINETICS

The primary route of exposure to DEHP is via inhalation; ingestion of DEHP from surface water and from foods and beverages packaged in polyvinyl chloride products can also occur. Dermal absorption of DEHP is also likely (EPA 1980).

DEHP and its metabolites are readily absorbed from the gastrointestinal tract, intraperitoneal cavity, and lungs. The absorption, distribution, and elimination of DEHP can be influenced by the vehicle in which it is administered (EPA 1980).

Absorbed DEHP is distributed to various organs and tissues in animals and humans, primarily to the lungs, heart, liver, kidney, gonads, and spleen. Excretion of DEHP occurs in urine and feces; excretion into bile also occurs and can increase the amount of DEHP (or metabolites) in the intestine (EPA 1980).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENICITY

DEHP is reported to be carcinogenic in rats and mice, causing increased incidences of hepatocellular carcinomas or neoplastic nodules following oral administration (NTP 1982).

MUTAGENICITY

The results of dominant lethal experiments in mice suggest that DEHP is mutagenic when injected intraperitoneally (Singh et al. 1974). However, most experiments conducted with microorganisms and mammalian cells have failed to demonstrate genotoxic activity (EPA 1980).

TERATOGENICITY/REPRODUCTIVE EFFECTS

Singh et al. (1972) reported dose-related gross or skeletal abnormalities in mouse fetuses from pregnant females injected intraperitoneally with 5 or 10 ml/kg DEHP. Nikonorow et al. (1973) orally administered 0.34 and 1.70 g/kg/day DEHP to pregnant rats during the gestation period and reported a reduction in fetal weight and increased number of resorptions. Other reproductive effects, including testicular changes in mice and rats, have also been reported (NTP/IRLG 1982).

ACUTE/CHRONIC TOXICITY

DEHP appears to have a relatively low toxicity in experimental animals. The oral, intraperitoneal, and intravenous LD_{50} values reported for DEHP in rats are 31 g/kg, 30.7 g/kg, and 0.25 g/kg, respectively. DEHP is poorly absorbed through the skin, and no irritant response or sensitizing potential from dermal application has been note in experimental animals or humans (NIOSH 1985, NTP/IRLG 1982, EPA 980).

Chronic exposure to relatively high concentrations of DEHP in the diet can cause retardation of growth and increased liver and kidney weights in experimental animals (NTP/IRLG 1982, EPA 1980).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying EPA's criteria for evaluating the overall weight of evidence of carcinogenicity (EPA 1986a), DEHP would be most appropriately classified in Group B2--Possible Human Carcinogen. This category applies to agents for

which there is inadequate evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals.

Because DEHP has been identified as a hepatocarcinogen in B6C3F$_1$ mice and F344/N rats (NTP 1982), it is prudent to consider the potential carcinogenicity of this compound. Using the NTP (1982) data for male mice, EPA (1986b) recently calculated an oral cancer potency factor (q_1^*) of 6.86×10^{-4} $(mg/kg/day)^{-1}$.

More recently, EPA (1986b) recommended a reference dose (RfD) of 0.02 mg/kg/day for DEHP. This value was based on a study by Carpenter et al. (1953) in which guinea pigs were fed diets containing 0, 0.04, or 0.13% DEHP for 1 year. No treatment-related effects were observed on mortality, body weight, kidney weight, or on gross pathology or histopathology of kidney, liver, lung, spleen, or testes. Statistically significant increases in relative liver weights were observed in both groups of treated females (64 and 19 mg/kg/day). Using 19 mg/kg/day as the lowest-observed-adverse-effect level and a safety factor of 1,000, the RfD of 0.02 mg/kg/day was derived.

EPA (1980) derived an acceptable daily intake (ADI) for DEHP from the results of a chronic ingestion study in rats, guinea pigs, and dogs. Although details of this derivation were not presented, an ADI of 0.6 mg/kg/day (42 mg/day) was calculated. Based on this value, EPA recommended an ambient water quality criterion (AWQC) of 15 mg/liter for protection of human health from the toxic properties of DEHP ingested through water and contaminated aquatic organisms. The AWQC for exposure to DEHP in drinking water alone would be 21 mg/liter. The lower value of 15 mg/liter was used to back-calculate an acceptable intake chronic (AIC) of 0.86.

SUMMARY OF DEHP CRITERIA

EPA carcinogen classification	Group B2
Oral cancer potency factor (q_1^*)	6.86×10^{-4} $(mg/kg/day)^{-1}$
RfD	0.02 mg/kg/day

AWQC:

Ingestion of water and aquatic organisms	15 mg/liter
Ingestion of water	21 mg/liter

REFERENCES

CARPENTER, C.P., WEIL, C.S., and SMYTH, H.F. 1953. Chornic oral toxicity of di(2-ethylhexyl)phthalate for rats, guinea pigs and dogs. Arch. Indust. Hyg. Occup. Med. 8:219-226

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 40/5-80-067

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Guidelines for carcinogen risk assessment. Fed. Reg. 51:33992-34003 (September 24, 1986)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Verified Reference Doses of the U.S. EPA. ADI Work Group of the Risk Assessment Forum, Washington, D.C. January 1986. ECAO-CIN-475

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1985. Registry of Toxic Effects of Chemical Substances Data Base. Washington, D.C. July 1985

NATIONAL TOXICOLOGY PROGRAM/INTERAGENCY REGULATORY LIAISON GROUP (NTP/IRLG). 1982. The Conference on Phthalates. Environ. Health Perspect. 45:1-153

NIKONOROW, M., ET AL. 1973. Effect of orally administered plasticizers and polyvinyl chloride stabilizers in the rat. Toxicol. Appl. Pharmacol. 26:253 (As cited in EPA 1980)

SINGH, A.R., ET AL. 1972. Teratogenicity of phthalate esters in rats. J. Pharmacol. Sci. 611 (As cited in EPA 1980)

SINGH, A.R., ET AL. 1974. Mutagenic and antifertility sensitivities of mice to di-2-ethylhexylphthalate (DEHP) and dimethoxyethyl phthalate (DMED). Toxicol. Appl. Pharmacol. 29:35 (As cited in EPA 1980)

CADMIUM

INTRODUCTION

Cadmium is an element of the transitional metal series that occurs widely in nature, usually in lead or zinc ores. Elemental cadmium is insoluble in water, although many cadmium salts are quite soluble (EPA 1985 a,b). The general human population is exposed to cadmium in drinking water and food; cigarette smoke also contains high levels of cadmium. Additional inhalation exposure occurs in industrial settings (EPA 1985a,b).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

The toxicology of cadmium has been reviewed by Friberg et al. (1974), IARC (1976), and EPA (1980, 1981, 1985b,c). Injection of cadmium into laboratory animals results in injection-site sarcomas and testicular tumors of the Leydig cells (EPA 1981). A relationship between human exposure to cadmium and cancer of the prostate, lung, or kidney has been suggested by several epidemiological studies (Thun et al. 1985, EPA 1985b). Cadmium may impair DNA repair but has not been shown to be mutagenic. It is a well-documented animal teratogen.

Cadmium bioaccumulates in mammals, particularly in the kidney and liver (EPA 1981, 1985c). Epidemiological studies have revealed an association between nonmalignant pulmonary diseases and inhalation of cadmium. Renal tubular dysfunction, of which the first sign is proteinuria, occurs at lower levels of oral or inhalation exposure to cadmium and may be the primary defect responsible for the bone damage characteristic of Itai-Itai disease. It is also suspected that chronic exposure to cadmium produces hypertension, anemia, sensory loss (particularly smell), endocrine alterations, and immunosuppression in humans.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

INGESTION TOXICITY

The maximum contaminant level (MCL) established for cadmium by EPA in its national interim primary drinking water standards and the Ambient Water Quality Criterion for the protection of human health are both 10 ug/liter (EPA 1980, CFR 1984). EPA established the standard for cadmium on the basis of the "generally accepted" estimate of 200 ug/g wet weight of cadmium in the renal cortex as the critical concentration for renal toxicity. Friberg et al. (1974) estimated that daily ingestion of 250-350 ug cadmium over 50 years would result in such renal concentrations. Other more recent reviews suggest that 200 ug/day is an acceptable daily limit for cadmium intake.

The EPA Office of Drinking Water has recently promulgated a proposed Recommended Maximum Contaminant Level (RMCL) of 0.005 mg/liter. The proposed RMCL is based on the estimate of 200 ug/g wet weight of cadmium in the renal cortex as the critical concentration for renal toxicity (Freiberg et al. 1974) and a 25% contribution to daily exposure from drinking water. The proposed RMCL is comparable to the NAS and WHO guidelines of 0.005 mg/liter (EPA 1985a).

Aughey et al. (1984) reported that treatment of male Wistar rats for only 24 weeks with 50 mg/liter cadmium in their drinking water produced clear symptoms of nephritis when the accumulated renal concentration of cadmium was 60 ug/g wet weight. This last concentration is less than one-third the 200 ug/g wet weight identified as the critical level in humans.

Cadmium and some of its compounds are known to be carcinogenic in experimental animals exposed by injection or inhalation, but the carcinogenic effects are absent when cadmium is administered orally (EPA 1985b). It is not known which cadmium compounds are responsible for the inhalation carcinogenic effects in humans.

INHALATION TOXICITY

EPA (1981) has reviewed the health risks posed by airborne cadmium compounds in considerable detail, while studies on the carcinogenicity of cadmium compounds have been reviewed by the IARC (1976). Both reviews were updated by EPA (1985b), which also presented a risk assessment for exposure to airborne cadmium.

Applying the criteria described in EPA's Guidelines for Assessment of Carcinogenic Risk (EPA 1986), cadmium has been classified by EPA (1985a) on the basis of inhalation data in Group B1--Probable Human Carcinogen. This category applies to agents for which there is limited evidence from human studies and sufficient evidence from animal studies (EPA 1984).

The evidence that exposure to airborne cadmium compounds increases the risk of cancer in humans is characterized as limited (IARC 1982, EPA 1985b). Although several studies of exposed workers have suggested that airborne cadmium increases the risk of cancer of the lung or prostate, most of the results have been inconclusive because of small sample sizes, lack of statistical significance, confounding effects of other exposures, or other factors. The most recent study (Thun et al. 1985), however, showed a significant increase in the number of lung cancer deaths (16 observed versus 6.99 expected) among a group of cadmium smelter workers. Although this finding may be somewhat confounded by the effects of smoking and exposure to arsenic, EPA (1985b) concluded that neither of the latter was sufficient to explain the observed effect.

EPA (1985b) based its quantitative risk assessment for inhaled cadmium on the study by Thun et al. (1985). Very scanty information on exposure levels and incomplete information on exposure duration were used to compile a single average measure of cumulative exposure of the exposed workers. This was converted to a lifetime average exposure level. The extent of the deviations of the exposure estimates from the actual exposure is unknown. Use of the lifetime average exposure level in the risk calculations incorporates the questionable assumption that exposure to cadmium at any time

during life would lead to the same increase in lifetime risk of lung cancer. The data were then fitted to a model which assumed that the effect of exposure to cadmium would be to increase the background rate of lung cancer by a factor proportional to the lifetime average exposure level. The selection of this model was arbitrary, in that no information on the dose-response relationship was provided between exposure and induction of lung cancers, and excess risk is likely to be very conservative. The best estimate of the exposure and response of the exposed population gave rise to a "unit risk" estimate of 1.8×10^{-3} $(ug/m^3)^{-1}$. However, because of uncertainties in the estimates of both exposure and response, the range of uncertainties in the unit risk estimate was very wide, ranging from 4.3×10^{-6} to 3.8×10^{-2} $(ug/m^3)^{-1}$. The last figure was obtained by combining "worst case" estimates of all exposure and risk parameters, and probably represents a "worse than worst case" calculation.

Given the severe limitations of the available date, this quantitative risk assessment appears as reasonable as could be achieved. However, the estimate of "unit risk" is extremely uncertain and probably extremely conservative. The study itself is not regarded as fully conclusive in showing any increased cancer risk and at least part of this is attributable to confounding factors. The exposure estimates are very uncertain, the assumption of a linear exposure response relationship is arbitrary, and the extrapolation over a range of 5 orders of magnitude in dose is very tenuous. Thus, the estimate of "unit risk" presented by EPA (1985b) could be inaccurate and is likely to be conservative when applied to general population exposure.

Recent studies have firmly associated inhalation of airborne cadmium with lung cancer in animals. In a bioassay in which male Wistar rats were administered cadmium chloride aerosols 23 hours/day for 18 months at concentrations of 0, 12.5, 25, and 50 ug/m^3, Takenaka et al. (1983) found a highly significant dose-response relationship. The incidence of lung carcinomas was 0/38, 6/39, 20/38, and 25/35 in the four treatment groups. This marked response adds further support to the evidence on the carcinogenicity of inhaled cadmium and justifies the use of Thun et al.'s (1985)

data as the basis for risk assessments of airborne cadmium. Like the injection-site sarcomas and Leydigomas reported in animals (EPA 1985b), these lung carcinomas might be interpreted as carcinogenic responses in the directly exposed tissue only.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) recommends a time-weighted average Threshold Limit Value (TLV) of 0.05 mg/m^3 for all forms of cadmium dusts and salts and a ceiling limit of 0.05 mg/m^3 for cadmium oxide fume.

SUMMARY

Cadmium has not been demonstrated to be a systemic carcinogen, but it has been shown to be a highly potent pulmonary carcinogen (see Takenaka et al. 1983) and renal effects are associated with ingestion. For inhalation, the unit risk value of 1.8×10^{-3} $(ug/m^3)^{-1}$ is an appropriate basis for risk assessment. This unit risk was derived from epidemiological data and does not incorporate any conservative assumptions (except that the dose-response relationship is linear), but it is subject to a wide range of uncertainty. For ingestion, the calculations upon which the MCL and ambient water quality criterion are based are equally applicable to ingestion via soil or other media. Based on the assessment of Friberg et al. (1974), daily ingestion of 20 ug cadmium (in addition to background exposure via food) would contribute between 6% and 8% of the estimated critical daily intake of cadmium. This intake of 20 ug/day from other than normal dietary sources was intended to provide an ample margin of safety, but Aughey et al.'s (1984) study suggests that it may not be as conservative as originally intended. The EPA Office of Drinking Water has recently promulgated a proposed Recommended Maximum Contaminant Level of 5 ug/liter for cadmium (EPA 1985d). This value is based on a reference dose of 5×10^{-4} mg/kg/day after allowing for contribution to daily exposure by other routes.

Summary of Cadmium Criteria

EPA carcinogen classification	Group B1
Oral chronic allowable intake	5×10^{-4} mg/kg/day
Inhalation carcinogenic potency (q_1^*)	6.1 $(mg/kg/day)^{-1}$
Maximum Contaminant Level (MCL)	10 ug/liter
Proposed RMCL	5 ug/liter
EPA Drinking Water Health Advisories	
Lifetime Health Advisory (HA)	5 ug/liter
Longer-term HA	
Child	5 ug/liter
Adult	18 ug/liter
Shorter-term HA	
10-day HA (child)	8 ug/liter
One-day HA (child)	43 ug/liter
AWQC	
Ingestion of water and aquatic organisms	10 ug/liter
Freshwater aquatic life chronic toxicity	exp(0.7852(ln hardness)-3.490)

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

AUGHEY, E., FELL, G.S., SCOTT, R., and BLACK, M. 1984. Histopathology of early effects of oral cadmium in the rat kidney. Environ. Health Perspect. 54:153-161

CODE OF FEDERAL REGULATIONS (CFR). 1984. 40 CFR 141, Environmental Protection Agency, National Interim Primary Drinking Water Regulations. U.S. Government Printing Office, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1981. Health Assessment Document for Cadmium. Research Triangle Park, North Carolina. EPA 600/8-81-023.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms; proposed rule. 40 CFR Part 141. Fed. Reg. 50(219):46936-47025 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium. Addendum to the Health Assessment Document for Cadmium (May 1981; EPA/600/8-81/023). Office of Health and Environmental Assessment, Washington, D.C. June 1985. EPA 600/3-33-025F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985c. Drinking Water Criteria Document for Cadmium (Final Draft). Office of Drinking Water, Washington, D.C. April 1985. PB86 117934/AS

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985d. Water quality criteria; notice of final ambient water quality criteria documents. Fed. Reg. 50:30784-30796. (July 29, 1985)

EVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Guidelines for carcinogenic risk assessment. Fed. Reg. 51:33992-34003. (September 24)

FRIBERG, L.T., PISCATOR, M., and NORDBERG, G. 1974. Cadmium in the Environment. 2nd ed. CRC Press, Cleveland

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1976. IARC Monographs on the Carcinogenic Risk of Chemicals to Man. Vol. 11: Cadmium, Nickel, Some Epoxides, Miscellaneous Industrial Chemicals, and General Considerations on Volatile Anaesthetics. World Health Organization, Lyon, France. Pp. 39-74

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 29: Some Industrial Chemicals and Dyestuffs. World Health Organization, Lyon, France

TAKENAKA, S., OLDIGES, H., KOENIG, H., HOCHRAINER, D., and OBERDOERSTER, G. 1983. Carcinogenicity of cadmium chloride aerosols in W rats. JNCI 70:367-373

THUN, M.J., SCHNORR, T.M., SMITH, A.B., HALPERIN, W.E., and LEMEN, B.A. 1985. Mortality among a cohort of U.S. Cadmium production workers - An update. JNCI 74:325-333

CHROMIUM

INTRODUCTION

Chromium is a steel-grey lustrous metal with the atomic number 24 and an atomic weight of 52 g/mole. It can have a valence of 2, 3, or 6. Chromium is used in stainless and alloy steels, in corrosion-resistant products, in pigments, and as a tanning agent for leather.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Chromium is an essential nutrient for normal glucose metabolism, and low level exposure to chromium compounds has not been found to result in systemic toxicity. However, hexavalent chromium (Cr VI) compounds are strong oxidizing agents, a characteristic that explains much of their irritating and toxic properties (EPA 1980). Chronic inhalation of dust or air containing Cr VI in the form of chromic acid or as soluble salts may cause respiratory problems including irritation, perforated or ulcerated nasal septa, and decreased spirometric values. Studies of acute exposure of humans and animals using a variety of routes of administration have indicated that soluble Cr VI and trivalent chromium (Cr III) compounds can produce kidney and liver damage, although the dose levels employed were relatively high. From the evidence available from both human case reports and animal studies, it is uncertain whether the kidneys and liver may be target organs following chronic exposure to chromium compounds. Dermal exposure to chromic acid or chromium VI salts may cause contact dermatitis, sensitivity, and ulceration of the skin (EPA 1984a).

Epidemiological studies of worker populations have clearly established that inhaled Cr VI is a human carcinogen, with the respiratory passages and the lungs as target organs. Convincing evidence for carcinogenicity of Cr VI compounds in animals is not available, except for the induction of sarcomas at the sites of implantation or injection of certain chromium VI salts and chromium trioxide (EPA 1980, 1984a).

Neither chromium III or chromium IV compounds have been shown to be carcinogenic by the oral route. Rats administered chromium III oxide at doses up to 5% of their diet for two years had no increase in tumor rates over control animals (Ivankovic and Preussmann 1975 as cited in EPA 1984a). Dogs given Cr VI as potassium chromate at concentrations of 0.45 to 11.2 mg/liter in drinking water for 4 years had no observed adverse effects (Anwar et al. 1961 as cited in EPA 1984a).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

The health risks posed by airborne chromium III and chromium VI have been reviewed in considerable detail by EPA (1984b). Studies on the carcinogenicity of chromium compounds were earlier reviewed by the International Agency for Research on Cancer (1980). A risk assessment for exposure to airborne chromium VI was included in the health assessment document by EPA (1984b). The following summary is based in part on these reviews and risk assessments, augmented by review of key primary papers. However, Clement scientists were not able to review the unpublished study by Mancuso (1975) which served as the primary basis for EPA's risk assessment.

EPA (1984b) based its quantitative risk assessment for inhaled hexavalent chromium on an unpublished study by Mancuso (1975). This study, although limited, was judged to be more adequate for risk assessment than three other studies (conducted in Norway, Sweden, and the USSR) which suffered from deficiencies in measurement of exposure, statistical power, and reporting. Mancuso's study showed excess risks of lung cancer in workers exposed to chromates between 1931 and 1937 and followed until 1974. Lung cancer risks increased with duration of exposure and with age. Estimates of cumulative exposure to soluble, insoluble, and total chromium were derived from a single set of industrial hygiene measurements taken in 1949. Smoking habits of the workers were not determined or discussed.

To construct dose-response relationships, EPA (1984b) converted all exposure data into average lifetime ambient concentrations. This is equivalent to assuming that exposure to chromium at any time during life leads to

the same increase in lifetime risk of lung cancer; this assumption is questionable, especially in view of the fact that measures of exposure were available for only one year. The data were fitted to a dose-response relationship of the Weibull type, in which cancer risk is assumed to be proportional to age raised to the kth power and to a second order polynominal $(ad + bd^2)$ in dose d. Fitting the data to observed estimates of age-specific cancer risk and average exposure of the chromate workers, EPA estimated that k was approximately 1.9 and both the linear and quadratic terms in d were significant. At low doses, the term ad dominates the risk calculations, and for lifetime exposure the "unit risk" was calculated to be 1.2×10^{-2} $(ug/m^3)^{-1}$. However, because of small sample size (only 35 observed lung cancer deaths distributed over 9 age-exposure classes), the statistical uncertainty of this estimate was very high (standard deviation = 7 x mean). Expressed in terms of total intake, the cancer potency factor was calculated as 41 $(mg/kg/day)^{-1}$ (EPA 1984b).

Although Mancuso's unpublished study is not available for independent review, EPA's procedures in using the data appear reasonable. However, the results are subject to a wide range of statisitcal uncertainty, in addition to their incorporation of a number of assumptions that are uncertain and/or conservative. EPA suggested that the assumptions made about exposure and the failure to control for smoking might have led to overestimation of risk by a factor of up to 4. The assumption that risk would be proportional to average lifetime exposure introduces additional uncertainties. More important, the linear extrapolation of the dose-response relationship estimated for highly-exposed workers to predict risks of the general population exposure to concentrations about 10^5 times lower is likely to be very conservative. Finally, the unit risk estimate is directly applicable only to persons exposed to chromium compounds similar to those involved in the study by Mancuso. EPA (1984b) stated (without giving details) that trivalent and hexavalent chromium compounds were present in the ratio 6:1, so that the unit risk for hexavalent chromium might be 7 times higher than that estimated for total chromium. Overall, therefore, EPA's estimate of the unit risk for exposure to chromium in ambient air must be characterized as extremely uncertain (primarily because of statistical uncertainty and

uncertainty in measuring exposure) and probably very conservative (primarily because of the wide range of extrapolation, but also because of assumptions about exposure and failure to control for smoking).

Based on exposure via inhalation, the IARC (1982) classified Cr VI as Group 1, human carcinogen. The evidence for the carcinogenicity of Cr III compounds was judged to be inadequate. However, IARC (1980) concluded that the epidemiological data do not allow elucidation of the relative contributions to carcinogenic risk of Cr III or Cr VI or of soluble versus insoluble compounds. EPA classified inhaled Cr VI as Group A--Human Carcinogen and inhaled Cr III as Group D--Not Classified (EPA 1984b, 1984c). Ingested chromium compounds were classified as Group D (EPA 1984d).

Hexavalent chromium's potent carcinogenic effects when inhaled make calculation of subchronic or chronic allowable intakes by inhalation for non-carcinogenic endpoints of toxicity inappropriate (EPA 1984b). For trivalent chromium, a chronic allowable intake (AIC) by inhalation of 0.0051 mg/kg/day was calculated in the Health Effects Assessment for Trivalent Chromium (EPA 1984c). This number was derived from a TLV. The studies used for derivation of the TLV involved workers concomitantly exposed to other dusts and fumes (ACGIH 1986).

For oral exposure to Cr VI, a subchronic allowable intake (AIS) of 0.025 mg/kg/day was derived in the Health Effects Assessment for Hexavalent Chromium (EPA 1984b). The AIS was based on a one-year study in which rats were exposed to 0-25 mg/liter Cr VI as potassium chromate in drinking water. Increased tissue concentrations of chromium, but no adverse health effects were reported at the highest dose (Mackenzie et al. 1958 as cited in EPA 1984b). An oral chronic allowable intake (AIC) of 0.005 mg/kg/day was derived from the same study, with application of an additional safety factor of 5 to adjust for less than lifetime exposure (EPA 1984b).

For oral exposure to Cr III, a subchronic allowable intake of 14 mg/kg/day was established based on a study by Ivankovic and Preussmann (1975 as cited in EPA 1984c). Rats were fed Cr III as chromium oxide at 2% and

5% of the diet for 90 days. A NOAEL of 5% Cr III was suggested, which corresponds to 1,399 mg/kg/day based on measured food consumption and body weight. Slightly depressed spleen and liver weights were reported.

For chronic oral exposure to Cr III, the EPA RfD Workgroup identified a risk reference dose (RfD) of 1 mg/kg/day based on the Ivankovic and Preussman (1975) study. Rats were fed chromic oxide baked in bread at dietary levels of 0, 1, 2, or 5%, 5 days/week for 600 feedings (840 total days). No effects due to chromic oxide treatment were observed at any dose levels.

The interim MCL for total chromium under the National Interim Primary Drinking Water Regulations is 0.05 mg/liter. A proposed Recommended Maximum Contaminant Level (RMCL) of 0.12 mg/liter is proposed for total chromium, based on an Adjusted Allowable Daily Intake (AADI) of 0.17 mg/liter with data on human exposure by other routes factored in (0.10 mg/day via the diet and 0 mg/day via air) (EPA 1985).

SUMMARY

Chromium is an essential micronutrient and is not toxic in trace quantities. High levels of soluble Cr VI and Cr III produce kidney and liver damage. Chronic inhalation exposure may lead to respiratory system damage. Epidemiological studies of workers exposed to Cr VI via inhalation demonstrate that it is a human carcinogen. EPA's (1984b) quantitative risk assessment is based upon a single study of a cohort of workers exposed over a six year period and followed for approximately 40 years. Smoking habits were not considered in the study. Assumptions and extrapolations included in the calculations lead to a very wide range of uncertainty in the risk factor. The relative carcinogenic potency of Cr III has not been demonstrated. A Risk Reference Dose (RfD) of 1 mg/kg/day has been established for oral exposure to Cr III. An oral AIC of 0.005 mg/kg/day was derived for Cr VI.

Summary of Chromium Criteria

EPA carcinogen classification (inhalation of Cr VI only)	Group A
Inhalation carcinogenic potency factor (q_1*)	41 $(mg/kg/day)^{-1}$
Oral AIC (Cr VI)	0.005 mg/kg/day
Oral risk reference dose (Cr III)	1 mg/kg/day
Maximum Contaminant Level (MCL) (total Cr)	0.05 mg/liter
Proposed RMCL (total Cr)	0.12 mg/liter

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. Cincinnati, Ohio.

ANWAR, R.A., LONGHAM, R.F., HOPPERT, C.A., ALFREDSON, B.V., and BYERRUM, R.V. 1961. Chronic toxicity studies III. Chronic toxicity of cadmium and chromium in dogs. Arch. Environ. Health 3:456-460

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Chromium. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-035

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Assessment Document for Chromium. Environmental Criteria and Assessment Office. Research Triangle Park, N.C. EPA 600/8-83-014F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Health Effects Assessment for Hexavalent Chromium. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-019

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984c. Health Effects Assessment for Trivalent Chromium. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-035

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984d. Draft Health Advisory for Chromium. Office of Drinking Water. Washington, D.C. September 30 draft

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms; proposed rule. Fed. Reg. 50:46936- 47025. (November 13)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Second Set of Verified Reference Doses. Memo from the Office of Health and Environmental Assessment to the RfD Workshop. May 14, 1986

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Suppl. 4. World Health Organization, Lyon, France. Pp. 133-135

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1980. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 23: Some metals and metallic compounds. World Health Organization, Lyon, France. Pp. 205-323

IVANKOVIC, S. and PREUSSMANN, R. 1975. Absence of toxic and carcinogenic effects after administration of high doses of chromic oxide pigment in subacute and long-term feeding experiments in rats. Food Cosmet. Toxicol. 13:347-351

MACKENZIE, R.D., BYERRUM, R.V., DECKER C.F., HOPPERT, C.A., and LONGHAM F.L. 1958. Chronic Toxicity Studies II. Hexavalent and trivalent chromium administered in drinking water to rats. Arch. Ind. Health 18:232-234

MANCUSO, T.F. 1975. International Conference on Heavy Metals in the Environment. Toronto, Canada. October 27-31 (cited in EPA 1984c).

CYANIDE

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Hydrogen cyanide and its simple salts, such as sodium cyanide, are highly toxic by all routes (ACGIH 1986, EPA 1980). Many reports are available regarding acute poisonings in humans. Hydrogen cyanide vapor is irritating at very low concentrations, is considered dangerous at 20 ppm (20 mg/m^3), and is fatal at concentrations of 100 ppm (100 mg/m^3) for 1 hour. NIOSH (1976) noted reports of chronic poisoning resulting in fatigue, weariness, and other subjective symptoms in workers, but these findings have been disputed by other investigators. Chronic exposure to low levels of cyanide salts has been reported to cause enlargement of the thyroid gland in humans, apparently due to inefficient elimination of the cyanide metabolite thiocyanate. NIOSH (1976) concluded that there was no evidence of carcinogenicity, mutagenicity, or teratogenicity for cyanides. Cyanide has been shown to produce chromosome breaks in a bean plant, Vicia faba. Because of its mechanism of action, inhibition of the electron transport system in oxidative phosphorylation, cyanide is acutely toxic to almost all forms of life.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

EPA (1986) calculated a reference dose (RfD) for cyanide. The RfD was based on a study by Howard and Hanzal (1955) in which rats were maintained for 104 weeks on a diet containing 76 or 190 mg/kg of cyanide. From data reported on food consumption and body weight, daily doses were estimated at 3.6 to 7.5 mg/kg/day for the male rats and 4.6 or 10.8 mg/kg/day for the female rats. No clinical or histological effects were observed at either dose level. Using the highest no-observed-adverse-effect level (NOAEL) OF 10.8 mg/kg/day and a safety factor of 500 (10 for species extrapolation, 10 for sensitive members of the population, and 5 to compensate for the apparent tolerance to cyanide when it is ingested in food rather than in water), an RfD of 0.02 mg/kg/day was derived.

EPA (1985) recommended a lifetime health advisory of 750 ug/liter for exposure to cyanide in drinking water. It was assumed that exposed individuals weigh 70 kg and ingest 2 liters of water per day. The difference between this value and a drinking water criteria based on the RfD noted above (700 ug/liter) is due to rounding error.

The ambient water quality criterion (AWQC) for cyanide is 200 ug/liter (EPA 1980).

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) has recommended a time-weighted average threshold limit value of 5 mg/m^3 for occupational exposure to cyanide.

SUMMARY OF CYANIDE CRITERIA

RfD	0.02 mg/kg/day
Lifetime HA	750 ug/liter
AWQC	200 ug/liter

REFERENCES

AMERICAN COUNCIL OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Qualty Criteria for Cyanides. Office of Water Regulations and Standards, Washington, D.C. October 1980. EPA 440/5-80-037

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Draft Health Advisory for Cyanide. Office of Drinking Water, Washington, D.C. September 30, 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Verified Reference Doses (RfDs) of the U.S. EPA. ADI Work Group of the Risk Assessment Forum. ECAO-CIN-475. January 1986

HOWARD, J.W., and HANZAL, R.F. 1955. Chronic toxicity for rats of food treated with hydrogen cyanide. J. Agric. Food Chem. 3:325-329

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1976. Criteria for a Recommended Standard—Occupational Exposure to Hydrogen Cyanide and Cyanide Salts (NaCN, KCN, and $Ca(CN)_2$). Washington, D.C. DHEW Publication No. (NIOSH) 77-108

1,1-DICHLOROETHANE

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Limited information is available concerning the effects of 1,1-dichloroethane (1,1-DCA). The extent or rate of absorption of 1,1-DCA has not been determined, but based on its chemical properties, rapid gastrointestinal absorption and moderate absorption from inhalation are expected (EPA 1984a).

1,1-DCA is probably less toxic than the 1,2-isomer (EPA 1980). At one time, the compound was used as an anesthetic, but it induced cardiac arrhythmias and its use was discontinued. It is probable that human exposure to sufficiently high levels of 1,1-DCA would cause central nervous system depression and respiratory tract and skin irritation, since many of the chlorinated aliphatics cause these effects (Parker at al. 1979). However, no dose-response data concerning these effects are available. Renal damage was observed in cats exposed by inhalation in a subchronic study (Hoffman et al. 1971). Inhalation exposure of pregnant rats to high doses of 1,1-DCA (6,000 ppm) retarded fetal development (Schwetz et al. 1974).

A carcinogenicity bioassay of 1,1-DCA was limited by poor survival of both treatment and control groups, and the physical conditions of the treated animals was markedly stressed. A dose-related increase in the number of tumors was not observed by the Fisher exact test, but the data suggest that the compound may have carcinogenic properties (NCI 1978).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying the criteria for evaluating the weight of evidence for carcinogenicity proposed by EPA's Carcinogen Assessment Group (EPA 1984b), 1,1-DCA is most appropriataly classified inGroup D--Not Classified (EPA 1984a). This category applies to agents for which there is inadequate human and animal evidence of carcinogenicity.

EPA derived health-based criteria for subchronic and chronic exposure to 1,1-dichlorethane in its Health Effects Assessment for this compound (EPA 1984a). The oral subchronic acceptable intake (AIS) and chronic acceptable intake (AIC) were calculated from the Hoffman et al. (1971) study in which cats exposed to 500 ppm 1,1-DCA for 26 weeks had no observed effects, but cats exposed to 500 ppm for 13 weeks and then 1,000 ppm for an additional 13 weeks suffered renal damage. The other mammalian species tested (rats, rabbits, and guinea pigs) had no observed adverse effects. The oral AIS is 1.2 mg/kg/day. The oral AIC of 0.12 mg/kg/day was derived from the AIS by applying an additional safety factor of 10 to compensate for less than lifetime exposure in the animal study (EPA 1984a).

The inhalation AIS of 1.38 mg/kg/day and the AIC of 0.14 mg/kg/day were also derived from the Hoffman et al. (1971) study (EPA 1984a).

EPA (1980) was unable to determine an ambient water quality criterion for humna health for 1,1-dichloroethane because of insufficient mammalian toxicological information.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) has recommended a time-weighted average threshold limit value of 200 ppm (810 mg/m^3) for occupational exposure to 1,1-dichloroethane.

SUMMARY OF 1,1-DICHLOROETHANE CRITERIA

EPA carcinogen classification	Group D
Oral AIS	1.2 mg/kg/day
Oral AIC	0.12 mg/kg/day
Inhalation AIS	1.38 mg/kg/day
Inhalation AIC	0.14 mg/kg/day

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for 1,1-Dichloroethane. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Effects Assessment for 1,1-Dichloroethane. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-027

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Proposed guidelines for carcinogen risk assessment. Fed. Reg. 49:46294-46301 (November 23, 1984)

HOFFMAN, H.T., BIRNSTIEL, H., and JOBST, P. 1971. The inhalation toxicity of 1,1 and 1,2-dichloroethane. Arch. Toxikol. 27:248-265 (As cited in EPA 1980)

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of 1,1-Dichloroethane for Possible Carcinogenicity. CAS No. 75-34-3. NCI Carcinogenesis Technical Report Series No. 66, Washington, D.C. DHEW Publication No. (NIH) 78-1316 (As cited in EPA 1984a)

PARKER, J.C., CASEY, G.E., and BAHLNON, L.J. 1979. NIOSH Current Intelligence Bulletin No. 27. Chloroethanes: Review of toxicity. Am. Ind. Hyg. Assoc. J. 40:A46-A60 (As cited in EPA 1980)

SCHWETZ, B.A., LEONG, B.K.J., and GEHRING, P.J. 1974. Embryo and fetotoxicity of inhaled carbon tetrachloride, 1,1-dichloroethane and methyl ethyl ketone. Toxicol. Appl. Pharmacol. 28:452-464 (As cited in EPA 1980)

1,2-DICHLOROETHANE

PHARMACOKINETICS

Data on the pharmacokinetics of 1,2-dichloroethane in humans are limited, but data from animal studies suggest that the chemical is rapidly absorbed following oral and inhalation exposure and after dermal contact with the liquid form of the compound (EPA 1985a). Reitz et al. (1982) reported that a 150 mg/kg bw peroral dose of radio-labelled 1,2-dichloroethane given to rats was completely absorbed 48 hours after administration, and Spreafico et al. (1978, 1979, 1980) reported 78% absorption in rats given a single oral dose of 25 mg/kg bw 1,2-dichloroethane in a water vehicle. Absorption rates of inhaled 1,2-dichloroethane have not been quantified, but because the chemical has a moderately high vapor pressure (80 mm Hg at 25°C) and a high blood-air partition coefficient (19.5), pulmonary absorption is probably rapid and complete (EPA 1984). Dermal absorption of 1,2-dichloroethane following vapor exposure is negligible, but direct skin application studies in mice (Tsuruta 1975, 1977) and guinea pigs (Jakobson et al. 1983) indicate that percutaneous absorption following direct liquid contact can be substantial and is linearly related to exposure duration.

After peroral or pulmonary absorption, 1,2-dichloroethane is distributed to all body tissues. The substance readily passes the blood-brain and placental barriers (EPA 1985a). In rats, the highest tissue concentrations following acute exposure are reached in the adipose tissue, followed by the blood and liver (Spreafico et al. 1978, 1977, 1980).

Studies in mice (Yllner 1971) and rats (Reitz et al. 1980, 1982) indicate that 1,2-dichloroethane is extensively metabolized, and the liver is the primary site of metabolic activity (EPA 1985a). 1,2-Dichloroethane is metabolized to 2-chloroacetaldehyde, S-(2-chloroethyl)-glutathione, and other putative reactive metabolites capable of covalent binding to cellular macromolecules (EPA 1985a). Metabolism appears to be dose related; percent metabolized decreases as dose increases (Yllner 1971, Reitz et al. 1980). Elimination of unmetabolized 1,2-dichloroethane is almost exclusively through the lungs (EPA 1985a).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENICITY

No epidemiological data are available to evaluate the carcinogenicity of 1,2-dichloroethane in humans. However, several chronic bioassays of 1,2-dichloroethane have been conducted in animals and some of these are outlined below.

In a study sponsored by the National Cancer Institute (NCI), groups of 50 male and 50 female 9-week-old Osborne-Mendel rats were administered technical grade 1,2-dichloroethane (>99% pure) in corn oil by gavage 5 days/week for 78 weeks (NCI 1978). Surviving animals were observed for up to 32 weeks following termination of treatment. The time-weighted average doses were 95 and 47 mg/kg bw/day for high- and low-dose males and females. A group of 20 male and 20 female rats received corn oil alone and were used as matched vehicle controls, and an additional group of 60 male and 60 female rats received corn oil and were used as pooled vehicle controls. (The group of matched vehicle controls was not considered in the statistical analyses of tumor incidences.) Mortality was increased in the high-dose group; all high-dose females were dead by week 15 of the post-treatment observation period, and all high-dose males died by week 23 of the observation period. All low-dose rats survived the 32-week post-treatment observation period. All treated and control animals were histologically examined. There was a statistically significant increase in total number of tumors in high-dose females when compared with pooled controls. Additionally, a statistically significant increase in the incidence of squamous-cell carcinomas of the forestomach in high-dose male rats and mammary gland adenocarcinomas in high-dose female rats was observed. Hemangiosarcomas occurred in some treated rats but not in any of the controls. Low-dose males and females showed higher incidences of hemangiosarcomas than high-dose animals. There was a statistically significant positive trend in the incidence of hemangiosarcomas in male and female rats as compared with pooled vehicle controls.

In a second oral-dosing study sponsored by NCI (1978), groups of 50 male and 50 female 5-week-old $B6C3F_1$ mice were administered technical grade (>99% pure) 1,2-dichloroethane in corn oil by gavage 5 days/week for 78 weeks, followed by a 12-week (females) or 13-week (males) period without treatment. The time-weighted doses were 195 and 299 mg/kg bw/day for high-dose males and females, respectively, and 97 and 149 mg/kg bw/day for low-dose males and females, respectively. A group of 60 male and 60 female mice received corn oil alone and served as pooled vehicle controls. Of the high-dose animals, 42% of the males survived until the end of the study, and 72% of the females died between weeks 60 and 80. In the low-dose group, 48% of the males survived more than 74 weeks, and 68% of the females survived until the end of the study. In the vehicle control groups, 55% of the males and 80% of the females survived until the end of the study. When compared with controls, the number of animals with tumors and the total number of tumors were significantly greater in both male and female high-dose mice and also in low-dose female mice.

Maltoni et al. (1980) exposed groups of 180 male and 180 female Sprague-Dawley rats and Swiss mice to 1,2-dichloroethane (>99% pure) vapor concentrations of 250-150 ppm, 50 ppm, 10 ppm, or 5 ppm, respectively, for 7 hours/day, 5 days/week for 78 weeks. (The initial 250 ppm concentration was reduced to 150 ppm after several days of exposure to the higher level elicited severe toxic effects.) Two groups of 180 rats per group served as controls for the rat study, and one group of 249 mice served as the control for the mice study. At the end of the treatment period, all animals were allowed to live until spontaneous death. A complete necropy was performed on all rats and mice. The results of the histopathological analyses revealed no statistically significant increases in the incidence of any specific type of tumor in treated rats or mice when compared with controls.

MUTAGENICITY

1,2-Dichloroethane has been shown to cause gene mutations in bacteria, plants, Drosophila, and cultured Chinese hamster ovary cells (EPA 1985a). The majority of the in vitro tests have reported marginal positive responses without metabolic activation and stronger positive responses with exogenous

hepatic metabolic activation, indicating that 1,2-dichloroethane is mutagenic by itself but that metabolites are more potent mutagens. In addition, 1,2-dichloroethane also has been observed to cause meiotic chromosomal nondisjunction in Drosophila and to alkylate DNA in several somatic tissues in the rat (EPA 1985a).

REPRODUCTIVE TOXICITY

Available data suggest that 1,2-dichloroethane does not adversely affect reproductive or developmental processes in experimental animals except at maternally toxic levels (EPA 1985a). However, the data are limited, and additional experimental and epidemiological studies are needed to conclusively demonstrate that this compound is not a human teratogen and does not produce adverse reproductive effects.

CHRONIC TOXICITY

Data on chronic toxicity of 1,2-dichloroethane in humans are limited (primarily) to foreign case reports and health surveys. Repeated exposure to 1,2-dichloroethane vapor in the work place has been reported to result in anorexia, nausea, vomiting, weakness and fatigue, nervousness, epigastric pain, and irritation of the respiratory tract and eyes (EPA 1984, 1985a). Kozik (1957) reported that workers in the Russian aircraft industry who were exposed for at least 4 years to approximately 10-15 ppm 1,2-dichloroethane experienced increased incidences of gastrointestinal disease, liver and gall bladder disease, and diseases of the muscles, tendons, and ganglia. Cetnarowicz (1959) reported dizziness, fatigue, drowsiness, nausea, and epigastric pain in Polish oil refinery workers exposed to 1,2-dichloroethane concentrations between 10 and 200 ppm for an unspecified period of time.

Chronic studies in animals also have revealed toxic effects following inhalation exposure. Spencer et al. (1951) exposed eight guinea pigs of both sexes, one female and two male rabbits, and two male monkeys to 1,2-dichloroethane vapor at concentrations of 400 and 100 ppm for 7 hours/day, 5 days/week for 6 months. In addition, 15 rats of both sexes and 8 guinea pigs of both sexes were similarly exposed to 200 ppm 1,2-dichloroethane for

151 and 180 7-hour periods, respectively. Exposure to 200 ppm 1,2-dichloroethane caused parenchymatous degeneration of the liver in guinea pigs. Severe toxic effects were found in all animals exposed to the 400-ppm concentration, but no toxic effects were observed in any animals exposed to the 100-ppm concentration.

Heppel et al. (1946) observed increased mortality and/or significant pathological changes in various organs of rats, rabbits, and guinea pigs exposed to 1,000 or 400 ppm of 1,2-dichloroethane 7 hours/day for 5 days/week but not in animals similarly exposed to concentrations of 100 ppm.

No pertinent data on the chronic oral toxicity of 1,2-dichloroethane in humans were located in the available literature, and limited reports of chronic oral exposure in laboratory animals were available. In the NCI (1978) bioassay previously discussed, groups of 50 male and 50 female Osborne-Mendel rats were treated by gavage with time-weighted average (TWA) 1,2-dichloroethane concentrations of 45 or 74 mg/kg bw/day, respectively, for 78 weeks and observed for up to an additional 32 weeks. Groups of 50 male and 50 female $B6C3F_1$ mice were treated by gavage with TWA doses of 195 or 97 mg/kg bw/day (male) or 299 or 149 mg/kg bw/day (female) 1,2-dichloroethane for 78 weeks, followed by a 12 to 13-week observation period. No significant dose-related body weight depression in rats of either sex was noted, but there was increased mortality, particularly in the high-dose groups, which appeared to be related to toxic rather than to carcinogenic effects. Mean body weight depression was noted as early as week 15 of the study in high-dose female mice but not in high- or low-dose males or low-dose females.

In a chronic feeding study, rats received diets containing 1,2-dichloroethane at concentrations of 0, 250, or 500 mg/kg fed for 2 years (Alumot et al. 1976). No biochemical or histopathological abnormalities attributable to 1,2-dichloroethane were observed. However, there was a widespread incidence of chronic respiratory disease and low survival rates. A lack of detailed data prevents establishment of the actual dose administered.

ACUTE TOXICITY

Effects of acute inhalation exposure in humans are similar to those observed in humans following chronic work-time exposure and include irritation of mucous membranes and the respiratory tract and central nervous system depression (EPA 1985a). Death may occur as a result of respiratory and circulatory failure. Pathologic examinations typically show congestion, degeneration, necrosis, and hemorrhagic lesions of most internal organs, including the liver, kidney, spleen, lungs, and respiratory and gastrointestinal tracts (EPA 1985a). Adverse effects caused by less extreme exposures were generally associated with the gastrointestinal and nervous systems.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying EPA's criteria for evaluating overall weight of evidence of carcinogenicity to humans, 1,2-dichloroethane has been classified in Group B2 as a probable carcinogen in humans. This category applies to agents for which there is inadequate evidence of carcinogenicity from human studies and sufficient evidence of carcinogenicity from animal studies.

EPA (1985a) derived a carcinogenic potency factor (q_1^*) for ingestion based on the incidences of hemangiosarcomas in Osborne-Mendel male rats observed in the NCI (1978) gavage study outlined previously. The final time-weighted average doses in male and female rats were 95 and 47 mg/kg/day for the high- and low-dose groups, respectively. Animals were observed for up to an additional 32 weeks before terminal sacrifice. Based on the hemangiosarcoma response in male rats using a time-to-death adjustment and an adjusted dose derived from the metabolism/kinetic evaluation, EPA used the multistage model to estimate an upperbound carcinogenic potency factor of 9.1×10^{-2} $(mg/kg/day)^{-1}$ for 1,2-dichloroethane.

EPA (1980) also based is ambient water quality criterion for 1,2-dichloroethane on the incidences of hemangiosarcomas observed in male rats in the NCI (1978) study previously discussed. However, because a somewhat

different approach was used, a carcinogenic potency factor of 3.697×10^{-2} $(mg/kg/day)^{-1}$ was derived. The resulting water concentration to keep the lifetime excess cancer risk below 10^{-6} was 0.94 ug/liter. This value assumes ingestion of contaminated water and aquatic organisms (e.g., fish) from the contaminated water. Without consumption of contaminated aquatic organisms, a lifetime cancer risk of 10^{-6} would be associated with ingestion of water containing 0.95 ug/liter of 1,2-dichloroethane.

Another carcinogenic potency factor, also based on the NCI (1978) hemangiosarcoma data for male rats, was reported in the EPA Health Effects Assessment for 1,2-dichloroethane (EPA 1984); the reported value was 6.9×10^{-2} $(mg/kg/day)^{-1}$.

The EPA Office of Drinking Water developed a longer-term health advisory (HA) for 1,2-dichloroethane based upon the results of two chronic inhalation studies (Heppel et al. 1946, Spencer et al. 1951) and one subchronic inhalation study (Hofmann et al. 1971) in which various animal species were exposed to 1,2-dichloroethane. In these studies, exposure to air containing 100 ppm 1,2-dichloroethane for 6-7 hours/day, 5 days/week for up to 8 months resulted in no mortality and no adverse effects on behavior, growth, organ function, or blood chemistry. Similar exposures to 400-500 ppm 1,2-dichloroethane resulted in high mortality and various pathological effects. The 100-ppm level (405 mg/m^3) was used as the no-observable-adverse-effect level (NOAEL) for the determination of the HA. The HAs derived for a 10-kg child consuming 1 liter of water per day and for a 70-kg adult consuming 2 liters of water per day are 740 ug/liter and 2,800 mg/liter, respectively.

EPA (1985b) recently published under the Safe Drinking Water Act, a proposed maximum contaminant level (MCL) of 5 ug/liter for 1,2-dichloroethane. The proposed MCL was determined based upon consideration of best available technology for removal of 1,2-dichloroethane from drinking water and upon the lowest achievable detection level for 1,2-dichloroethane by routine laboratory operating conditions within specified limits of precision and accuracy.

SUMMARY OF 1,2-DICHLOROETHANE CRITERIA

EPA carcinogen classification	Group B2
Oral carcinogenic potency factor (q_1*)	9.1×10^{-2} (mg/kg/day)$^{-1}$
Longer-term HA:	
Child	740 ug/liter
Adult	2,600 mg/liter
Proposed MCL	5 ug/liter
AWQC (concentration associated with a 10^{-6} lifetime cancer risk:	
Ingestion of water and aquatic organisms	0.94 ug/liter
Ingestion of water only	0.95 ug/liter

REFERENCES

ALUMOT, E., NACHTOMI, E., MANDEL, E., HOLSTEIN, P., BONDI, A., and HERTZBERG, M. 1976. Tolerance and acceptable daily intake of chlorinated fumigants in the rat diet. Food Cosmet. Toxicol. 14:105-110 (As cited in EPA 1985c)

CETNAROWICZ, J. 1959. Experimental and clinical studies on effects of dichloroethane. Folia. Med. Cracov. 1:169-192 (As cited in EPA 1985a).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Chlorinated Ethanes. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for 1,2-Dichloroethane. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-002

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Health Assessment Document for 1,2-Dichloroethane. Office of Health and Environmental Assessment, Washington, D.C. September 1985. EPA 600/8-84-006F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulations; volatile synthetic organic chemicals, final rule. Fed. Reg. 50:46880-46901 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985c. Draft Health Advisory for 1,2-Dichloroethane. Office of Drinking Water, Washington, D.C.

HEPPEL, L.A., NEAL, P.A., PERRIN, T.L., ENDICOTT, K.M., and PORTERFIELD, V.T. 1946. Toxicology of 1,2-dichloroethane. V. Effects of daily inhalations. J. Ind. Hyg. Toxicol. 28:113-120 (As cited in EPA 1985a).

HOFMANN, H.T., BIRNSTEIL, H., and JOBST, P. 1971. Zur inhalation toxicatat von 1,1- and 1,2-dichloroathan. Arch. Toxikol. 27:248-265 (As cited in EPA 1985a)

JAKOBSON, I., WAHLBERT, J.E., HOLMBERT, B., and JOHANSSON, G. 1983. Uptake via the blood and elimination of 10 organic solvents following epicutaneous exposure of anesthetized guinea pigs. Toxicol. Appl. Pharmacol. 63:181-187 (As cited in EPA 1985a)

KOZIK, I. 1957. Problems of occupational hygiene in the use of dichloroethane in the aviation industry. Gig. Tr. Prof. Zabol. 1:31-38 (As cited in EPA 1985a)

MALTONI, C., VALGIMIGLI, L., and SCARNATO, C. 1980. Long-term carcinogenic bioassays on ethylene dichloride administered by inhalation by rats and mice. In Ames, B.N., Infante, P., Ritz, R., eds. Ethylene Dichloride: A Potential Health Risk? Banbury Report No. 5. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York. Pp. 3-33 (As cited in EPA 1985a)

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of 1,2-Dichloroethane for Possible Carcinogenicity. NCI Carcinogenesis Technical Report Series No. 55. Washington, D.C. DHEW (NIH) Publication No. 78-1361 (As cited in EPA 1985a)

REITZ, R.H., FOX, T.R., DOMORADZKI, J.Y., QUAST, J.F., LANGVARDT, P., and WATANABE, P.G. 1980. Pharmacokinetic and macromolecular interactions of ethylene dichloride: Comparison of oral and inhalation exposures. In Ames, B.N., Infante, P., and Reitz, R., eds. Ethylene Dichloride: A Potential Health Risk? Banbury Report No. 5. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York. Pp. 135-148 (As cited in EPA 1985a)

REITZ, R.H., FOX, T.R., RAMSEY, J.C., QUAST, J.F., LANGVARDT, P.W., and WATANABE, P.G. 1982. Pharmacokinetics and micromolecular interactions of ethylene dichloride in rats after inhalation or gavage. Toxicol. Appl. Pharmacol. 62:190-204 (As cited in EPA 1985a)

SPENCER, H.C., ROWE, V.K., ADAMS, E.M., McCOLLISTER, D.D., and IRISH, D.D. 1951. Vapor toxicity of ethylene dichloride determined by experiments on laboratory animals. Ind. Hyg. Occup. Med. 4:482 (As cited in EPA 1985a)

SPREAFICO, F., ZUCCATO, E., MARCURI, F., ET AL. 1978. Metabolism of 1,2-Dichloroethane in Experimental Animals. Report Nos. 1 and 2. Chemical Manufacturers Association, New York (As cited in EPA 1985a)

SPREAFICO, F., ZUCCATO, E., MARCURI, F., ET AL. 1979. Distribution and Metabolism of 1,2-Dichloroethane (EDC) in Experimental Animals. Report Nos. 3 and 4. Chemical Manufacturers Association, New York (As cited in EPA 1985a)

SPREAFICO, F., ZUCCATO, E., MARCURI, F., SIRONI, M., PAGLIALUNGA, S., MADONNA, M., and MUSSINI, E. 1980. Pharmacokinetics of ethylene dichloride in rats treated by different routes and its long-term inhalatory toxicity. In Ames, B.N., Infante, P., Reitz, R., eds. Ethylene Dichloride: A Potential Health Risk? Banbury Report No. 5. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York. Pp. 107-133 (As cited in EPA 1985a)

TSURUTA, H. 1975. Percutaneous absorption of organic solvents. 1. Comparative study of the in vivo percutaneous absorption of chlorinated solvents in mice. Ind. Health 13:27-236 (As cited in EPA 1985a)

TSURUTA, H. 1977. Percutaneous absorption of organic solvents. 2. A method for measuring the penetration rate of chlorinated solvents through excised rat skin. Ind. Health 15:3-4, 131-139 (As cited in EPA 1985a)

YLLNER, S. 1971. Metabolism of 1,2-dichloroethane-^{14}C in the mouse. Acta Pharmacol. Toxicol. 30:257-265 (As cited in EPA 1985a)

1,1-DICHLOROETHYLENE

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Several comprehensive reviews of the toxicology and potential health effects of 1,1-dichloroethylene have been prepared. The following summary is based primarily on information presented in three EPA documents. These include "Ambient Water Quality Criteria for Dichloroethylene" (EPA 1980), "Drinking Water Criteria Document for Dichloroethylenes" (EPA 1984a), and "Health Assessment Document for Vinylidene Chloride" (EPA 1985a).

1,1-Dichloroethylene is a central nervous system depressant and has been used as an anesthetic. Chronic low-level exposure to this substance may result in neurotoxicity, nephrotoxicity, hepatotoxicity, and cardiac arrhythmia. Inhalation or oral exposure of rats and rabbits has produced fetotoxicity and minor skeletal abnormalities, but at doses associated with some degree of maternal toxicity.

1,1-Dichloroethylene has been found to be a point mutagen in bacterial systems with metabolic activation, but dominant lethal studies in rats and mice were negative. The evidence for the potential of 1,1-dichloroethylene to act as a human germ-cell mutagen is currently regarded by EPA (1985a) as limited. Although this designation indicates that there are insufficient data to classify this evidence as sufficient or suggestive of potential germ-cell mutagenicity, the available data also do not permit classification of 1,1-dichloroethylene as a non-germ cell mutagen.

A number of laboratory studies have investigated the carcinogenic potential of 1,1-dichloroethylene. A significantly increased incidence of renal adenocarcinoma was observed in male mice treated by inhalation with 1,1-dichloroethylene in a study by Maltoni et al. (1985); female mice in the same study showed an equivocal increase in mammary adenocarcinomas. Other studies with mice, rats and hamsters have not shown increased tumor incidences after exposure by the oral or inhalation routes.

The effects of 1,1-dichloroethylene on humans have not been studied in large enough populations to provide meaningful results (EPA 1980, 1985a).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

The EPA concluded that the evidence for carcinogenicity of 1,1-dichloroethylene is inadequate in humans and limited in experimental animals. Applying the criteria for evaluating the overall weight of evidence of carcinogenicity to humans proposed by the EPA (1984b), this compound is most appropriately classified in Group C--Possible Human Carcinogen.

The carcinogenic potency for exposure by ingestion was derived by EPA (1985a) by estimating an upper-limit value from the negative data of a drinking water study by Quast et al. (1983) in Sprague-Dawley rats and a negative NTP (1982) gavage study in Fischer 344 rats and B6C3F1 mice. Quast et al. (1983) treated Sprague-Dawley rats with 50, 100, or 200 ppm 1,1-dichloroethylene in their drinking water for 2 years and observed only minimal hepatotoxicity. In the NTP bioassay (NTP 1982), no significant increases in tumor incidence were observed, but a maximum tolerated dose was apparently not achieved in the gavage treatment of F344 rats with 1 or 5 mg/kg and B6C3F1 mice with 2 or 1[illegible] mg/kg 5 times a week for 104 weeks. This approach assumes that a response occurs via ingestion, although there is no direct evidence that this is true. The highest upper-bound carcinogenic potency (q_1^*) estimate using these data was 0.58 $(mg/kg/day)^{-1}$.

The carcinogenic potency for exposure by inhalation reported by EPA (1985a) is based on an inhalation study in mice by Maltoni et al. (1985). Swiss mice of both sexes were exposed to 0, 10, or 25 ppm (0, 40, or 100 mg/m^3) 1,1-dichloroethylene for 4 hours/day, 4 to 5 days/week for 52 weeks, and were observed for a total of 121 weeks. The most marked finding was the significantly increased incidence of renal adenocarcinomas in the high-dose male mice. Using data from this study, calculation of the cancer risk estimates by the multistage model yielded an inhalation carcinogen potency of 1.16 $(mg/kg/day)^{-1}$.

Under the Safe Drinking Water Act, EPA has recently promulgated a final recommended maximum contaminant level (RMCL) and proposed a maximum contaminant level (MCL) of 7 ug/liter for 1,1-dichloroethylene in drinking water (EPA 1985b). RMCLs are nonenforceable health goals that are to be set at levels that would result in no known or anticipated adverse health effects with an adequate margin of safety. MCLs, which are enforceable standards when finalized, are EPA set as close as feasible to RMCLs after consideration of treatment and analytical technologies, costs, and other factors.

In setting the 7 ug/liter RMCL for 1,1-dichloroethylene, EPA determined that limited but insufficient evidence of carcinogenicity exists for this compound. Accordingly, it was classified in Regulatory Category II--equivocal evidence of carcinogenicity. RMCLs for Category II contaminants are set based on chronic toxicity data with an additional margin of safety or on lifetime risk calculations if chronic toxicity data are not available. For 1,1-dichloroethylene, EPA set the RMCL based on chronic toxicity data, primarily liver effects, obtained from the Quast et al. (1983) study in rats. An adjusted acceptable daily intake (AADI) of 350 ug/liter was derived from these data. By applying an extra safety factor of 10 to account for the possible carcinogenicity of this compound and assuming a 20% contribution to exposure from drinking water, the final RMCL of 7 ug/liter was determined (EPA 1984, EPA 1985b).

The EPA Office of Drinking Water developed one-day, longer-term, and lifetime health advisories (HAs) for 1,1-dichloroethylene (EPA 1985c). The one-day HA derived for a 10-kg child is 1,000 ug/liter. This HA was based on a study in which adult rats received single doses of 100, 300, or 500 mg/kg 1,1-dichloroethylene in corn oil (Jenkins et al. 1972). A lowest-observed-adverse-effect level (LOAEL) of 100 mg/kg for liver enzyme changes was identified. The longer-term HA was based on a 90-day study in which rats received 0, 50, 100, or 200 ppm 1,1-dichloroethylene in their drinking water (Rampy et al. 1977). This study identified a no-observed-adverse effect level (NOAEL) of 100 ppm (a decreased kidney-body weight ratio in males was observed at 50 ppm); increased cytoplasmic vacuolization of hepatocytes was observed in both sexes at 200 ppm. Based on

the NOAEL of 100 ppm, a longer-term HA was derived for a 10-kg child consuming 1 liter of water per day and a 70-kg adult consuming 2 liters of water per day. These values are 1,000 ug/liter and 3,500 ug/liter, respectively. EPA noted that the longer-term HA of 1,000 ug/liter for a 10-kg child would also be appropriate for protection over a ten-day period.

A lifetime HA of 70 ug/liter was based on the Quast et al. (1983) study cited above. A LOAEL of 100 ppm was identified based upon a trend towards increased fatty deposition in the liver. A relative source contribution of 20% for drinking water was included in this value. A risk reference dose (RfD) of 0.01 mg/kg/day was determined by EPA from data presented in this study. An RfD is an estimate of daily exposure which appears to be without appreciable risk of deleterious noncarcinogenic effects over a lifetime of exposure. This value corresponds approximately to the oral reference dose (RfD) of 0.009 mg/kg/day (EPA 1986) also recommended by EPA and based on the Quast et al. (1983) study.

EPA (1980) determined an ambient water quality criteria (AWQC) of 0.06 ug/liter for 1,1-dichloroethylene, which corresponds to a 10^{-6} excess lifetime cancer risk.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) has recommended a time-weighted average threshold limit value of 5 ppm (20 mg/m^3) for occupational exposure to 1,1-dichloroethylene.

SUMMARY OF 1,1-DICHLOROETHYLENE CRITERIA

EPA carcinogen classification	Group C
Oral carcinogenic potency factor (q_1^*)	0.58 $(mg/kg/day)^{-1}$
Inhalation carcinogenic potency factor (q_1^*)	1.16 $(mg/kg/day)^{-1}$
Final MCL/proposed MCL	7 ug/liter
One-day HA (child)	1,000 ug/liter
Ten-day HA (child)	1,000 ug/liter

Longer-term HA	
Child	1,000 ug/liter
Adult	3,500 ug/liter
Lifetime HA	70 ug/liter
RfD	0.009 mg/kg/day
AWQC corresponding to a 10^{-6} excess lifetime cancer risk	0.06 ug/liter

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5Th ed. Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Dichloroethylenes. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-041

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Drinking Water Criteria Document for Dichloroethylenes. Office of Drinking Water, Washington, D.C. December 1984. PB 86-117785

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Proposed guidelines for carcinogen risk assessments; request for comments. Fed. Reg. 46291-46311 (November 23, 1984)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Health Assessment Document for Vinylidene Chloride. Final Report. Environmental Criteria and Assessment Office. Research Triangle Park, North Carolina. August 1985. EPA 600/8-83/031F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulations; volatile synthetic organic chemicals, final rule. Fed. Reg. 50:46880-46901 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985c Draft Health Advisory for 1,1-Dichloroethylene. Office of Drinking Water, Washington, D.C. September 30.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Verified Reference Doses (RfDs) of the EPA. Office of Health and Environmental Assessment, Washington, D.C. January 1986. ECAO-CIN-475

JENKINS, L.F., Jr., TRABULUS, M.J., and MURPHY, S.D. 1972. Biochemical effects of 1,1-dichlorethylene in rats: Comparison with carbon tetrachloride and 1,2-dichlorethylene. Toxicol. Appl. Pharmacol. 23:501-510

MALTONI, C., COTTI, G., MORISI, L., and CHIECO, P. 1977. Carcinogenicity bioassays and vinylidene chloride: Research plan and early results. Med. Lavoro 68:241-262

MALTONI, C., LEFEMINE, G., COTTI, G., CHIECO, P., and PATELLA, V. 1985. Archives of Research on Industrial Carcinogenesis. Volume III: Experimental Research on Vinylidine Chloride Carcinogenesis. Princeton Scientific Publishers, Princeton, New Jersey.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1982. Carcinogenesis Bioassay of Vinylidene Chloride in F344 Rats and B6C3F$_1$ Mice (Gavage Study). CAS No. 75-35-4. NTP Technical Report Series No. 55, Bethesda, Maryland. NIH Publication No. 82-1784

QUAST, J.F., HUMISTON, C.G., WADE, C.E., BALLARD, J., BEYER, J.E., SCHWEITZ, R.W., and NORRIS, J.M. 1983. A chronic toxicity and oncogenicity study in rats and subchronic toxicity in dogs on ingested vinylidene chloride. Fund. Appl. Toxicol. 3:55-62

RAMPY, L.W., QUAST, J.F., HUMISTON, C.G., BLAMER, M.F., AND SCHWEITZ, B.A. 1977. Interim results of two-year toxicological studies in rats of vinylidene chloride incorporated in the drinking water or administered by repeated inhalation. Environ. Health Perspect. 21:33-43

trans-1,2-DICHLOROETHYLENE

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Information on the health effects of trans-1,2-dichloroethylene is limited. Freundt et al. (1977) exposed female rats by inhalation to 200 ppm trans-1,2-dichlorothylene for 8 hours per day, 5 days per week for 1, 2, 8, or 16 weeks and noted progressive damage to the lungs and progressive fatty degeneration of the liver.

trans-1,2-Dichloroethylene was not mutagenic in the Ames assay and did not induce chromosomal aberrations in mouse bone marrow cells (Cerna and Kypenova 1977). Galli et al. (1982) reported that it was not mutagenic when tested in a diploid strain (D7) of Saccharomyces cerevisiae with and without metabolic activation. Finally, trans-1,2-dichloroethylene was not mutagenic in the Escherichia coli K12 assay system (Greim et al. 1975).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

According to EPA's Proposed Guidelines for Carcinogenic Risk Assessment (EPA 1984a), trans-1,2-dichloroethylene has been classified in Group D--Not Classified (EPA 1985). This category applies to agents for which there is inadequate evidence of carcinogenicity from animals studies.

EPA (1980) was unable to derive ambient water quality criteria for trans-1,2-dichloroethylene because of lack of data.

The EPA opinion concerning protection of human health from the toxic effects of trans-1,2-dichloroethylene in drinking water is summarized in proposed national primary drinking water regulations for synthetic organic chemicals (EPA 1985). Because compound-specific information concerning toxicity of the 1,2-dichloroethylenes is not adequate, EPA has chosen to derive longer-term (1- to 12-year) and lifetime exposure criteria for both the cis- and trans-isomers from data on 1,1-dichloroethylene, a compound with similar structure (a geometric isomer of 1,2-dichloroethylene) and similar noncarcinogenic toxic

end points. Derivation of these criteria are documented in detail in the Drinking Water Criteria Document for Dichloroethylenes (EPA 1984b). Available information from shorter-term exposures to all three compounds also suggests that the noncarcinogenic toxicity induced by the 1,2-isomers is likely to be no more severe than that of 1,1-dichloroethylene. Accordingly, EPA derived longer-term draft health advisories for both cis- and trans-1,2-dichloroethylene of 1,000 and 3,500 ug/liter for children and adults, respectively, from data on 1,1-dichloroethylene.

EPA also derived an acceptable daily intake (ADI) for lifetime exposure based on the results of a 2-year chronic toxicity/oncogenicity study (Quast et al. 1983) in which rats received 0, 50, 100, or 200 mg/liter 1,1-dichloroethylene in drinking water. For the highest dose group, significant microscopic liver changes were seen in animals of both sexes, and minimal hepatocellular swelling and fatty changes were seen in female rats at all dose levels. At 100 ppm (10 mg/kg mean daily dose in males), a trend toward an increase in focal fatty changes in the liver was observed in males. No exposure-related changes were seen in males at the low dose. An ADI of 0.01 mg/kg/day for a 70-kg person was determined using a lowest-observed-adverse effect level (LOAEL) of 10 mg/kg/day and an uncertainty factor of 1,000, since a no-observed-adverse effect level (NOAEL) was not identified. The corresponding adjusted acceptable daily intake (AADI) for a 70-kg person drinking 2 liters of water per day is 350 ug/liter. EPA proposed an RMCL of 70 ug/liter for both cis- and trans-1,2-dichloroethylene based on the AADI of 350 ug/liter, assuming 20% of the exposure is via drinking water. Based on consideration of currently available data, EPA's proposed recommended guidelines for longer-term and lifetime exposure appear adequate for protection of the health of exposed individuals.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) has recommended a time-weighted average threshold limit value of 200 ppm (790 mg/m^3) for occupational exposure to trans-1,2-dichloroethylene.

DI-n-BUTYL-PHTHALATE

INTRODUCTION

Di-n-butyl phthalate (DBP) is a phthalate ester commonly used as a plasticizer and in the production of polyvinyl chloride (PVC) resins (EPA 1980).

PHARMACOKINETICS

The phthalic acid esters and/or their metabolites are readily absorbed from the gastrointestinal tract, intraperitoneal cavity, and the lungs and distributed to various organs and tissues. The esters are excreted primarily in urine and feces (EPA 1980).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

There are no reports that DBP is carcinogenic in animals or humans. However, DBP was reported to be mutagenic in a bacterial test system (Seed 1982). Reduced fetal weight, resorptions, and dose-related musculoskeletal abnormalities were observed among fetuses from rats and mice exposed to very high doses of DBP during gestation (Shiota and Nishimura 1982).

The acute toxicity for laboratory animals by most routes of administration is very low. Oral, inhalation, and intraperitoneal LD_{50} values of 8,000 mg/kg, 7,900 mg/m^3, and 3,050 mg/kg, respectively, have been reported for the rat (EPA 1980). Smith (1953) fed male Sprague-Dawley rats diets containing 0, 0.01, 0.05, 0.25 and 1.25% di-n-butyl phthalate for 1 year. At the highest di-n-butyl phthalate concentration, one-half of all rats died during the first week of exposure.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying the carcinogen risk guidelines established by the Carcinogen Assessment Group of EPA (EPA 1986a), di-n-butyl phthalate is most

appropriately classified in Group D--Not Classified. This category applies to agents for which there is no evidence of carcinogenicity.

EPA (1986b) calculated a risk reference dose (RfD) of 0.1 mg/kg/day for di-n-butyl phthalate based on a study by Smith (1953) in which male Sprague-Dawley rats fed diets containing 0.25% (125 mg/kg/day) di-n-butyl phthalate did not exhibit any effects on gross pathology or hematology.

EPA (1980) also determined an ambient water quality criterion (AWQC) of 34.1 mg/liter for the protection of human health from the toxic properties of di-n-butyl phthalate ingested through water.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) has recommended a time-weighted average threshold limit value (TLV) of 5 mg/m^3 for occupational exposure to di-n-butyl phthalate.

SUMMARY OF DI-n-BUTYL PHTHALATE

EPA carcinogen classification	Group D
RfD	0.1 mg/kg/day
AWQC	34.1 mg/liter

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Washington, D.C. October 1980. EPA 440/5-80-067

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Guidelines for carcinogen risk assessment. Fed. Reg. 51:33992-34002 (September 24, 1986)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Verified Reference Doses (RfDs) of the U.S. EPA. Prepared by the ADI Group of the Risk Reference Forum, Washington, D.C. January 1986. CAO-CIN-475

SEED, J.L. 1982. Mutagenic activity of phthalate esters in bacterial liquid suspension assays. Environ. Health Perspect. 45:111-114

SHIOTA, K., and NISHIMURA, H. 1982. Teratogenicity of di(2-ethyhexyl)-phthalate (DEHP) and di-n-butyl phthalate (DBP) in mice. Environ. Health Perspect. 45:65-70

SMITH, C.C. 1953. Toxicity of butyl stearate, dibutyl sebacate, dibutyl phthalate, and methoxyethyl oleate. Arch. Ind. Hyg. 7:310 (As cited in EPA 1980)

ETHYLBENZENE

INTRODUCTION

Ethylbenzene (EB) is a clear, colorless, flammable liquid found in gasoline. It is used as a solvent and in the manufacture of styrene and acetophenone. Ethylbenzene is also a constituent of asphalt and naptha.

PHARMACOKINETICS

Ethylbenzene absorbed via inhalation was found to be distributed throughout the body in Harlan-Wistar rats. However, the highest levels were detected in the kidney, lung, adipose tissue, digestive tract, and liver (Chin et al. 1980a). Data regarding the absorption of ethylbenzene from the gastrointestinal tract following oral ingestion were not located in the available literature. The elimination of ethylbenzene is primarily through urinary excretion of metabolites (EPA 1984).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENICITY

Pertinent data on the carcinogenic potential of ethylbenzene were not identified in the available literature.

MUTAGENICITY

Ethylbenzene was found to be nonmutagenic in Salmonella typhimurium strains TA98, TA100, TA1535, and TA1537 tested with and without metabolic activation (Florin et al. 1980). Pure ethylbenzene did not increase the frequency of recessive lethals in the Drosophila recessive lethal test (Donner et al. 1979).

TERATOGENICITY/REPRODUCTIVE EFFECTS

Hardin et al. (1981) found that ethylbenzene did not elicit embryo toxicity, teratogenicity, or maternal toxicity in New Zealand white rabbits exposed for 6-7 hours/day at 100 or 1,000 ppm (435 or 4,348 mg/m^3) during gestation days 1-24. However, female Wistar or Sprague-Dawley rats exposed to 1,000 ppm ethylbenzene for 6-7 hours/day during gestation days 1-19 had increased liver, kidney, and spleen weights indicating maternal toxicity.

ACUTE/CHRONIC TOXICITY

Wolf et al. (1956) administered a single oral doses of ethylbenzene (in olive oil or corn oil solution) by stomach tube to male and female Wistar-derived rats to determine the acute toxicity of ethylbenzene. The reported LD_{50} for both sexes was 3.5 g/kg bw; systemic toxic effects occurred primarily in the liver and kidney.

Wolf et al. (1956) also administered ethylbenzene in olive oil by stomach tube 5 days/week for 6 months to groups of 10 female Wistar rats. Doses of 468 and 680 mg/kg caused increased liver and kidney weights and cloudiness and swelling of hepatocytes and renal tubular epithelium. No effects were observed in rats exposed to 13.6 and 136 mg/kg/day.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying the criteria in EPA's guidelines for assessment of carcinogenic risk (EPA 1986a), ethylbenzene is most appropriately classified in Group D--Not Classified. This category applies to agents for which there is inadequate evidence of carcinogenicity.

EPA (1985b) derived one-day health advisories (HAs) for ethylbenzene based on a study by Bordodej and Bordodejova (1970) in which no adverse health effects were reported in humans exposed via inhalation for 8 hours to 100 ppm (435 mg/m^3) ethylbenzene. The one-day HAs for a 10-kg child and 70-kg adult

are 73 mg/liter and 21 mg/liter, respectively. Because of a lack of appropriate exposure duration data, EPA (1985b) derived the ten-day HAs from the one-day HAs by dividing the one-day HAs by ten. The resulting ten-day HAs for a 10-kg child and 70-kg adult are 2.1 mg/liter and 7.3 mg/liter, respectively. EPA (1985b) derived a lifetime health advisory of 3.4 mg/liter from the Wolf et al. (1956) study in which rats were treated orally at four doses of ethylbenzene 5 days/week for 6 months exhibited no adverse effects at 136 mg/kg/day. Based on the lifetime health advisory of 3.4 mg/liter and assuming 20 percent of exposure to ethylbenzene would be via drinking water, EPA (1985c) proposed a recommended maximum contaminant level (RMCL) of 0.68 mg/liter.

EPA (1986b) determined a reference dose (RfD) of 0.1 mg/kg/day for oral exposure to ethylbenzene based on the Wolf et al. (1956) data using the no-observed-adverse-effect level (NOAEL) of 136 mg/kg/day

EPA (1980b) has established an ambient water quality criterion (AWQC) of 1.4 mg/liter for the protection of human health from the toxic properties of ethylbenzene ingested through water and contaminated aquatic organisms.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) has recommended a time-weighted average threshold limit value of 100 ppm (435 mg/m^3) for occupational exposure to ethylbenzene.

SUMMARY OF ETHYLBENZENE CRITERIA

EPA carcinogen classification	Group D
One-day HA:	
Adult	73 mg/liter
Child	21 mg/liter
Ten-day HA:	
Adult	7.3 mg/liter
Child	2.1 mg/liter
Lifetime HA	3.4 mg/liter
Proposed RMCL	0.68 mg/liter

RfD	0.1 mg/kg/day
AWQC	1.4 mg/liter

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

BORDODEJ, Z., and BORDODEJEVA, E. 1970. Biotransformation of ethylbenzene, styrene and alpha-methylstyrene in man. Am. Ind. Hyg. Assoc. J. 31:206-209 (As cited in EPA 1985b)

CHIN, B.H., McKELVEY, J.A., TYLER, T.R., CALISTI, L.J., KOZBELT, S.J., and SULLIVAN, L.J. 1980a. Absorption, distribution and excretion of ethylbenzene, ethylcyclohexane and methyl ethylbenzene isomers in rats. Bull. Environ. Contam. Toxicol. 24:477-483 (As cited in EPA 1985a)

DONNER, M., MAKI-PAAKKANEN, J., NORPPA, N., SORSA, M., and VAINIO, H. 1979. Genetic toxicology of xylenes. Mutat. Res. 74:171-172 (As cited in EPA 1985a)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980b. Ambient Water Quality Criteria for Ethylbenzene. Office of Water Regulations and Standards, Washington, D.C. October 1980. EPA 440/5-80-048

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Ethylbenzene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-008

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document for Ethylbenzene (Final Draft). Office of Drinking Water, Washington, D.C. March 1985. EPA 600/X-84-163-1

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Draft Health Advisory for Ethylbenzene. Office of Drinking Water, Washington, D.C. September 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985c. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms. Fed. Reg. 50:46936-47022 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Guidelines for carcinogenic risk assessment. Fed. Reg. 51:33992-34003 (September 24, 1986)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Verified Reference Doses (RfDs) of the U.S. EPA. Office of Health and Environmental Assessment, Washington, D.C. January 1986. ECAO-CIN-475

FLORIN, I., RUTHBERG, L., CURVALL, M., and ENZELL, C.R. 1980. Screening of tobacco smoke constituents for mutagenicity using the Ames test. Toxicology 18:219-232 (As cited in EPA 1985a)

HARDIN, B.D., BOND, G.P., SIKOV, M.R., ANDREW, F.D., BELILES, R.P., and NIEMEIER, R.W. 1981. Testing of selected workplace chemicals for teratogenic potential. Scand. J. Work Environ. Health 7:66-75 (As cited in EPA 1985a)

WOLF, M.A., ROWE, V.K., McCOLLISTER, D.D., HOLLINGSWORTH, R.L., and OYEN, F. 1956. Toxicoloigcal studies of certain alkylated benzenes and benzene. Arch. Ind. Health 14:387-398 (As cited in EPA 1985a)

HEXACHLOROBENZENE

INTRODUCTION

Hexachlorobenzene is a synthetic organic compound that is produced during the synthesis of several chlorinated compounds. At one time, hexachlorobenzene was used as a fungicide, but this use has been discontinued (EPA 1985a).

PHARMACOKINETICS

No information was present in the available literature on hexachlorobenzene absorption from the lungs or skin (EPA 1985a). Absorption of hexachlorobenzene from the gastrointestinal tract appears to depend on the vehicle used during test material administration (EPA 1985a). Eighty percent of the dose is absorbed when hexachlorobenzene is administered in olive oil; less than 20% of the dose is absorbed when hexachlorobenzene is administered in an aqueous solution (EPA 1985a, 1984a). Intestinal absorption of hexachlorobenzene occurs mainly via the lymphatic system (IARC 1979). Hexachlorobenzene is distributed to tissues that have a high lipid content (EPA 1985a). The adipose tissue accumulates the greatest concentrations of hexachlorobenzene in all of the species studied (EPA 1985a). Bone marrow and skin also accumulate large amounts of hexachlorobenzene (EPA 1985a). Hexachlorobenzene is metabolized to lesser chlorinated benzenes and chlorinated phenols which are excreted in the urine as glucuronide and glutathione conjugates together with small amounts of unchanged hexachlorobenzene (EPA 1985a). The excretion of hexachlorobenzene occurs primarily as the parent compound through the feces (EPA 1985a).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENICITY

Cabral et al. (1977) fed diets containing 0, 50, 100, or 200 ppm hexachlorobenzene to Syrian golden hamsters for life (EPA 1984a). These diets contained 4, 8, or 16 mg/kg/day hexachlorobenzene and resulted in significant

increases in the incidences of hepatomas and hemangioendothelioma of the livers of exposed male and female rats (EPA 1984a). The incidence of hepatoma was 47% for both sexes at 4-5 mg/kg/day (EPA 1984a). The incidences of hemangioendothelioma were 20% in males at 8 mg/kg/day and 12% in females at 16 mg/kg/day (EPA 1984a).

Liver hepatomas were also produced in both sexes of Swiss mice administered hexachlorobenzene for 106 weeks (EPA 1984a). At 24 mg/kg/day, the incidences were 34% for females and 16% for males (EPA 1984a).

Liver tumors (hepatomas or hepatocellular carcinomas) were found in three studies which included three different strains of rats: Agus, Wistar, and Sprague-Dawley (EPA 1985a). These tumors were induced at doses between 1.5 and 8 mg/kg/day (EPA 1985a). Smith and Cabral (1980) demonstrated an increased incidence of liver cell tumors in female MRC Wistar and Agus rats exposed to dietary levels of 100 ppm hexachlorobenzene for 75 to 95 weeks (EPA 1984a). Liver tumors were observed in rats exposed to 75 or 150 ppm hexachlorobenzene in the diet for up to 2 years (EPA 1984a). Arnold (1983) observed hepatocellular carcinomas in male and female Sprague-Dawley rats exposed to hexachlorobenzene in utero, during lactation, and in the diet for the remainder of their lifetimes (130 weeks) (EPA 1985a).

The data on hexachlorobenzene provide sufficient evidence of the carcinogenicity of hexachlorobenzene, since there were increased incidences of malignant tumors of the livers in two species (hemangioendothelioma in hamsters and hepatocellular carcinoma in rats) as well as reports of hepatomas in mice, rats and hamsters (EPA 1985a).

MUTAGENICITY

Hexachlorobenzene was not mutagenic when tested in the Salmonella typhimurium histidine reversion assay with and without metabolic activation (EPA 1985b). It did not induce dominant lethal effects in male rats (EPA 1985a). Hexachlorobenzene was found to be mutagenic in a yeast assay, Saccharomyces cerevisiae at a concentration of 100 ppm (EPA 1985a).

TERATOGENICITY/REPRODUCTIVE EFFECTS

Hexachlorobenzene is fetotoxic and produces some teratogenic effects in experimental animals (IARC 1979). Fetotoxic effects include elevated liver weights in rats and hepatomegaly and reduced survival in cats (EPA 1985a). Hexachlorobenzene given to pregnant mice was found to produce cleft palates and renal agenesis in exposed (concentrations unspecified) pups (EPA 1985a).

ACUTE/CHRONIC EFFECTS

The acute oral toxicity of hexachlorobenzene has been found to be low (EPA 1985a). The following LD_{50} values have been reported in the literature: rats, 3,500-10,000 mg/kg; rabbits, 2,600 mg/kg; cats, 1,700 mg/kg; and mice, 4,000 mg/kg (EPA 1985b).

Subchronic oral toxicity studies conducted in several mammalian species indicate a significant increase in liver and kidney weights (EPA 1985a). Histological changes have been identified in the livers of exposed animals (i.e., irregular shaped and moderately enlarged liver mitochondria and increases in the size of the centrilobular hepatocytes) (EPA 1985b).

Chronic oral toxicity studies revealed similar effects to those seen in the subchronic studies (EPA 1985a). In addition, hexachlorobenzene is associated with increases in mortality and with lesions of the liver and kidney (EPA 1985a). Other effects reported include multiple alopecia and scabbing, associated with neurological effects in rats, mice and dogs (EPA 1985a). Increased porphyrin levels in the liver and urine have been reported for all species studied except the dog (EPA 1985a).

The exposure of humans to hexachlorobenzene (0.05 to 0.2 g/day) in Turkey from 1955-1959 resulted in hexachlorobenzene-induced porphyria turcica (EPA 1985a). This disease is manifested by disturbed porphyrin metabolism, cutaneous lesions and hyperpigmentation (EPA 1985a). Follow-up studies conducted with patients 20 to 25 years after the onset of porphyria showed that a few patients still had active porphyria, whereas 50% exhibited

hyperpigmentation and scarring as well as other dermatologic, neurological and skeletal features of hexachlorobenzene toxicity (EPA 1985a).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying EPA's criteria for evaluating the overall weight of evidence of carcinogenicity to humans (EPA 1984b), hexachlorobenzene has been classified by EPA in Group B2--Probable Human Carcinogen. This category applies to agents for which there is inadequate evidence of carcinogenicity from human studies and sufficient evidence from animal studies (EPA 1984b).

EPA (1984c) derived a carcinogenic potency factor (q_1^*) of $1.7\ (mg/kg/day)^{-1}$ based on the incidence of hepatocellular carcinoma in female rats fed diets containing hexachlorobenzene for up to 2 years (EPA 1984a). The concentration in drinking water corresponding to a 10^{-6} excess lifetime cancer risk is 0.02 ug/liter (EPA 1984a).

The EPA Office of Drinking Water developed longer-term health advisories (HAs) of 0.175 mg/liter for a 70 kg adult and 0.050 mg/liter for a 10 kg child (EPA 1985b). The HAs are based on the study by Kuiper-Goodman et al. (1977) in which male and female Charles River rats were fed hexachlorobenzene at 0.5, 2.0, 8.0, or 32.0 mg/kg bw/day for 15 weeks. Female rats were found to be more susceptible to hexachlorobenzene, as indicated by all parameters studied, and a no-observed-adverse-effect level (NOAEL) of 0.5 mg/kg/day was concluded by the authors. At higher doses there was an increase in liver porphyrin levels in females and an increase in the size of centrilobular hepatocytes along with depletion of hepatocellular marker enzymes.

The EPA Office of Drinking Water also developed a drinking water equivalent level (DWEL) for noncarcinogenic end points of toxicity over a lifetime of exposure (EPA 1985b). A drinking water equivalent level of 0.028 mg/liter was derived for a 70-kg adult drinking 2 liters of water per day. The derivation of the DWEL was based on a 130-week study in which male and female Sprague-Dawley rats were exposed to hexachlorobenzene *in utero*, during lactation, and in the diet for the remainder of their lifetimes (EPA 1985b). A NOAEL of 1.6 mg/kg (0.08 mg/kg bw/day) was identified. At higher dietary

levels, there was an increase in the incidence of hepatic centrilobular basophilic chromogenesis. EPA (1985b) estimated the excess cancer risk that would result if exposure were to occur at the DWEL over a lifetime. The estimated excess cancer risk associated with lifetime exposure to drinking water containing hexachlorobenzene at 0.028 mg/liter is approximately 1×10^{-3}. This estimate represents the upper 95% confidence limit from extrapolations prepared by EPA's Carcinogen Assessment Group using the linearized, multistage model.

EPA (1980) has set ambient water quality criteria (AWQC) for hexachlorobenzene of 7.2, 0.72, and 0.072 mg/liter corresponding to cancer risks of 10^{-5}, 10^{-6}, and 10^{-7}, respectively, assuming 70-kg humans consume 2 liters water and 6.5 g fish and shell fish.

SUMMARY OF HEXACHLOROBENZENE CRITERIA

EPA carcinogen classification	Group B
Oral carcinogenic potency factor	1.7 $(mg/kg/day)^{-1}$
Longer-term HA:	
Adult	0.175 mg/liter
Child	0.050 mg/liter
DWEL	0.028 mg/liter
AWQC (corresponding to a 10^{-6} excess lifetime cancer risk	0.720 mg/liter

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th. ed. Cincinnati, Ohio

CABRAL, J.R.P., SHUBIK, P., MOLLNER, T., and RAITANO, F. 1977. Carcinogenic activity of hexachlorobenzene in hamsters. Nature 269:510-511

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Chlorinated Benzenes. Environmental Criteria and Assessment Office, Cincinnati, Ohio. EPA 440/5-80-028

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Effects Assessment for Hexachlorobutadiene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1986. EPA 540/1-86-017

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Proposed guidelines for carcinogen risk assessment; request for comments. Fed. Reg. 49:46294-46301 (November 23, 1984)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984c. Health Effects Assessment for Hexachlorobutadiene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-053

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document for Hexachlorobenzene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. ECAO-CIN-424. (Final Draft)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Draft Hexachlorobenzene Health Advisory. Office of Drinking Water. Washington, D.C., September 1985

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Vols. 19 and 20. World Health Organization, Lyon, France

KUIPER-GOODMAN, T., GRANT, D.L., MOODIE, C.A., KORSRUD, G.O., and MUNRO, I.C. 1977. Subacute toxicity of hexachlorobenzene in the rat. Toxicol. Appl. Pharmacol. 40:519-549

SMITH, A.G., and CABRAL, J. 1980. Liver cell tumors in rats fed hexachlorobenzene. Cancer Lett. 11(2):169-172

APPENDIX PHE-2

TOXICITY PROFILES OF SELECTED INDICATOR CHEMICALS

PHENOL

INTRODUCTION

Phenol is a high volume industrial chemical that is widely used as an intermediate in the manufacture of other chemicals and is a by-product of combustion and some industrial processes. It is also produced by biological processes, e.g., microbial metabolism in the gut and metabolism of certain drugs in vivo (EPA 1980). It is a component of several over-the-counter medicines for both topical and oral administration.

PHARMACOKINETICS

Phenol is readily absorbed through the gut, by inhalation, and percutaneously. Following absorption, the compound is rapidly distributed systemically. The majority of phenol and its metabolites are excreted rapidly in the urine (EPA 1980).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

The National Cancer Institute (NCI 1980) conducted a subchronic (90-day) study and a carcinogenesis bioassay of phenol administered in drinking water to F344/N rats and B6C3F$_1$ mice. In the subchronic study, doses of 100, 300, 1,000, 2,000, and 10,000 ppm phenol were not associated with mortality or histological changes; rats and mice of both sexes receiving 10,000 ppm consumed less water than animals in the control or other treatment groups and their body weights decreased. In the 105-week carcinogenesis bioassay, no treatment-related mortality, tumors, or nonneoplastic lesions were reported in rats or mice ingesting water containing 2,500 or 5,000 ppm phenol. A dose-related depression in mean body weight, which was related to decreased water consumption, occurred in both species of rodents.

Phenol exhibited tumor-promoting activity in the mouse skin painting system following initiation with 9,10-dimethyl-1,2-benzanthracene (DMBA) or benzo(a)pyrene (BaP), and it exhibited cutaneous carcinogenic activity in a

sensitive mouse strain when applied at concentrations that produced repeated skin damage (EPA 1980).

There are no data in the literature on the carcinogenicity of inhaled phenol in experimental animals or humans. The compound is mutagenic in several systems, but not in others (NCI 1980). There are no data on its teratogenicity (EPA 1984a).

Signs of acute phenol toxicity in humans and experimental animals are central nervous system depression, collapse, coma, cardiac arrest, and death. Acutely toxic doses can also cause extensive necrosis at the site of exposure (eyes, skin, oropharynx) (EPA 1980).

In a subchronic oral (gavage) study in rats (Dow Chemical Co. 1976), 0.1 g/kg phenol induced "slight liver changes and slight to moderate kidney damage" in animals. However, lack of study details (numbers of animals, incidence figures, specific lesions) in the 1980 EPA document make these results unreliable for interpreting the toxic changes. Subchronic inhalation studies conducted by Deichmann and Witherup (1944) in guinea pigs, rabbits, and rats were inadequately designed (no control groups). Therefore, caution should be used in interpreting the pulmonary, myocardial, renal, and hepatic damage as compound induced. The results of other subchronic inhalation studies are difficult to interpret based on the information in secondary sources (EPA 1980, 1984a).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying the criteria for overall weight of evidence of carcinogenecity proposed by the Carcinogen Assesment Group of EPA (EPA 1984b), phenol has been classified in Group D--Not Classified (EPA 1984a). This category applies to agents for which there are no data available regarding carcinogenicity in humans or experimental animals.

A reference dose (RfD) of 0.1 mg/kg/day for ingestion of phenol was based on the Dow Chemical Co. (1976) subchronic rat study. The ambient water quality criterion (AWQC) of 3.5 mg/liter/day for drinking water was extrapolated from

this study. The AWQC based on organoleptic properties was established at 0.3 mg/liter (EPA 1980).

An inhalation acceptable intake chronic (AIC) of 1.4 mg/person/day was recommended by EPA (1984a) based on the threshold limit value of 19 mg/m^3 phenol established by the American Conference of Governmental Industrial Hygienists (ACGIH 1983).

SUMMARY OF PHENOL CRITERIA

EPA Carcinogen Classification	Group D
Reference dose (RfD)	0.1 mg/kg/day
AIC (inhalation)	1.4 mg/person/day
AWQC	3.5 mg/liter

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1983. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1983-1984. Cincinnati, Ohio (As cited in EPA 1984a)

DEICHMANN, W.B., and WITHERUP, S. 1944. Phenol Studies. VI. The acute and comparative toxicity of phenol and o-, m-, and p-cresols for experimental animals. J. Pharmacol. Exp. Ther. 80:233 (As cited in EPA 1980)

DOW CHEMICAL CO. 1976. References and Literature Review Pertaining to Toxicological Properties of Phenol. Toxicol. Res. Lab. (unpublished manuscript) (As cited in EPA 1980)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Phenol. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-066

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Effects Assessment for Phenol. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-007

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Proposed guidelines for carcinogen risk assessment. Fed. Reg. 49:46294-46301 (November 23, 1984)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Verified Reference Doses of the EPA. ADI work Group of the Risk Assessment Forum, Washington, D.C. January 1986. ECAO-CIN-475

NATIONAL CANCER INSTITUTE (NCI). 1980. Bioassay of Phenol for Possible Carcinogenicity. CAS No. 108-95-2. NCI Carcinogenesis Technical Report Series No. 203. Washington, D.C. USDHHS Publication No. (NTP) 80-15

POLYCHLORINATED BIPHENYLS (PCBs)

INTRODUCTION

PCBs are complex mixtures of chlorinated biphenyls. The commercial PCB mixtures that were manufactured in the United States were given the trade name of "Aroclor." Aroclors are distinguished by a four-digit number (for example, Aroclor 1260). The last two digits in the Aroclor 1200 series represent the average percentage by weight of chlorine in the product.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

PHARMACOKINETICS

Absorption

PCBs are readily absorbed through the gastrointestinal (G.I.) tract and somewhat less readily through the skin. G.I. absorption in rats is greater than 90% for a wide range of isomers (Albro and Fishbein 1972). Dermal absorption of Aroclor 1242 in a benzene/hexane solution was 15-34% in monkeys (Wester et al. 1983). PCBs are presumably readily absorbed from the lungs, but few data are available that experimentally define the extent of absorption after inhalation (EPA 1985a).

Metabolism and Excretion

Absorbed PCBs in the blood are distributed initially to the liver, but within a few days the liver concentrations are greatly reduced as redistribution occurs. Less chlorinated chlorobiphenyl isomers are metabolized, but chlorobiphenyl isomers with higher percentages of chlorine are very lipophilic and are redistributed to adipose tissue, skin, and other organs (Matthews 1983). The major metabolic products of PCBs are phenolic derivatives or dihydrodiols.

Sequestering of PCBs in adipose tissue isolates them from metabolically active liver enzymes and greatly retards their clearance from the body. PCB metabolites are excreted in the urine and bile, apparently exclusively in the conjugated form. Excretion in urine is more prominent for less chlorinated chlorobiphenyl isomers while bile becomes significant for more highly chlorinated compounds (Safe 1980). Excretion of PCBs also has been reported in milk (Deichmann 1981).

Enzyme Induction

Like other organochlorine compounds, PCBs can cause marked induction of microsomal enzymes. Microsomal mixed-function enzymes metabolize a large variety of lipophilic compounds. In general, the intensity of induction increases with increasing chlorination of the mixture of PCBs and is dose-related. The enhanced activity of hepatic enzymes induced by PCBs can persist for a considerable period of time after dosing (NRCC 1978).

NONCARCINOGENIC HEALTH EFFECTS

PCBs have a number of documented toxic effects on humans and other mammals; noncarcinogenic effects are summarized in the following subsections.

Health Effects Observed in Humans

In considering the health effects of PCB exposure observed in humans, it is important to note that PCBs are often contaminated with highly toxic impurities, particularly polychlorinated dibenzofurans (PCDFs). As the effects due to PCDFs versus PCBs have not been separated in most human studies and because the two cause similar effects, the following discussion refers to commercial mixtures. The reader should recognize that at least some of the reported effects may be due to the PCDF impurities.

Dermatitis and chloracne (a disfiguring and long-term skin disease) have been the most prominent and consistent findings in studies of occupational exposure to PCBs. Interpretation of these data is complicated by the

difficulty of diagnosing chloracne and the uncertainties of blood PCB determinations. Reports of both chloracne and other PCB-related skin effects have generally been associated with exposures to more highly chlorinated PCB mixtures containing 42% chlorine or more (Chase et al. 1982, Emmett et al. 1983, Maroni et al. 1981a,b, Fischbein et al. 1979). Smith et al. (1982) reported no PCB-related skin effects in workers exposed to high levels of Aroclor 1016, which is less chlorinated.

Several studies examining liver function in exposed humans have reported disturbances in blood levels of liver enzymes. Maroni et al. (1981b), Chase et al. (1982), Smith et al. (1982), and Emmett et al. (1983) found statistically significant associations between blood PCB levels and elevated levels of GGTP, SGOT, or both. There is no evidence that a "no-effect" level exists for these effects, since correlations were found in individuals with low mean blood PCB levels.

Reproductive outcomes of women exposed to PCBs from high consumption of PCB-contaminated fish from Lake Michigan were compared to births from women who reported no such exposure (Fein et al. 1984a,b, Jacobson et al. 1983, Jacobson et al. 1984). Reduced birth weights, slow weight gain, reduced gestational ages, and behavioral deficits in infants were reported in a methodologically sound study. The study did not, however, rigorously establish that the causative factor was exposure to PCBs rather than other contaminants present in Lake Michigan fish.

Health Effects Reported from Animal Studies

Based on the published literature, reproductive, hepatic, and immunotoxic effects appear to be the most sensitive end points of PCB toxicity in non-rodent species, and the liver appears to be the most sensitive target organ for toxicity in rodents.

PCBs are not highly toxic when given as a single oral dose to mammals (Kimbrough et al. 1978), PCBs and would be classified as only slightly toxic based on acute oral toxicity (Hodge and Sterner 1949). The more significant

toxic effects of PCBs are observed on repeated exposure over a period of time.

The most consistent pathological changes occurring in mammals other than monkeys and mink after PCB exposure are in the liver (EPA 1985a). Fatty deposits and increased liver sizes are commonly reported. Individual hepatocytes may appear foamy and vacuolated. Cellular necrosis can occur at high doses, and the location of lesions is often centrilobular (Sleight 1983).

Comparative studies have shown that Aroclor 1254 is usually the most hepatotoxic mixture. In rats, Aroclor 1260 was significantly less toxic to the liver than Aroclor 1254 (Kimbrough et al. 1972). Aroclors 1248, 1242, and 1016 are progressively less toxic than Aroclor 1254 (Koller 1977, Burse et al. 1974).

A skin syndrome similar to the effects seen in humans has been reported in monkeys. Barsotti et al. (1976) and Allen et al. (1979) fed diets containing 2.5 and 5 ppm Aroclor 1248 to rhesus monkeys for 7 months. Weight loss, followed by hair loss, acne of the face and neck, eyelid edema and erythema, and keratinization of hair follicles were reported. McNulty et al. (1980) reported similar effects in rhesus monkeys exposed to Aroclor 1242 and individual chlorobiphenyl isomers. These authors indicated that swelling of the eyelids, resulting from dilation of the Meibomian glands is the most sensitive indicator of chlorinated aromatic hydrocarbon poisoning of monkeys.

Reproductive effects of PCBs have been demonstrated in animals, but there appear to be major differences in the susceptibility of different species. Mink and monkeys appear to be relatively sensitive; mice, rats, and rabbits appear relatively insensitive.

Dietary administration of PCBs for 9 months prior to whelping caused lowered reproductive success in mink. Feeding Aroclor 1254 at 2 ppm or Aroclor 1243 at 5 ppm was detrimental to reproduction. Aroclor 1016 at concentrations

as high as 20 ppm did not lower the number of young born per female. The PCBs were embryotoxic to developing embryos, but spermatogenesis, oogenesis, and implantation were unaffected. Transfer of PCBs to the surviving pups through mother's milk was a significant contributing factor to offspring exposure (Ringer 1983).

Monkeys also appear to be sensitive to the reproductive effects of PCBs. Altered menstrual cycles, reduced breeding success, and lowered birth rates were reported in monkeys exposed to dietary levels of 2.5 and 5.0 ppm Aroclor 1248 for 6 to 7 months before breeding (Barsotti et al. 1976). When female monkeys were exposed to 0.5 or 1 ppm Aroclor 1248 (three times per week) for 7 months prior to breeding (Allen et al. 1979, Bowman et al. 1981), no toxic signs were observed in the mothers. The infants, however, had reduced birth weights, developed hyperpigmentation during nursing, and were marginally hyperactive at age 12 months. "No-effect" levels have not been demonstrated for reproductive effects in monkeys, since toxicity was observed at the lowest doses tested. For comparison, Linder et al. (1974) performed a two-generation study in rats and reported dose-related effects (reduced numbers of litters, litter-sizes and pup survivals, and pathological lesions in offspring) of Aroclor 1254 at dietary concentrations down to 20 ppm. Aroclor 1260 was not as toxic in the same experiment.

Immunotoxic and immunosuppresive effects have been reported in most experiments in which these end points have been investigated, and are among the more sensitive indicators of PCB exposure (EPA 1985a). Monkeys seem to be the most sensitive species; guinea pigs are relatively sensitive; and rabbits, rats and mice appear relatively insensitive. Rhesus monkeys chronically exposed to Aroclor 1248 exhibited atrophy or hypocellularity of the thymus, spleen, lymph nodes, and bone marrow; these effects were particularly marked in infants after chronic exposures of their mothers to diets containing 2.5 ppm Aroclor 1248 (Allen and Barsotti 1976, Allen et al. 1980). Reductions in cell-mediated immunity, humoral immunity, or both were reported in monkeys chronically exposed to diets containing 1.5 or 5 ppm Aroclor 1248 (Thomas and Hinsdill 1978), and in monkeys dosed with

100 or 400 ug/kg/day Aroclor 1254 (Truelove et al. 1982). Increased susceptibilities to infectious diseases in PCB-exposed monkeys have been reported by Allen et al. (1979), McNulty et al. (1980), and Barsotti et al. (1976).

CARCINOGENIC HEALTH EFFECTS

A number of studies have suggested that PCB mixtures are capable of increasing the frequency of tumors in animals exposed to the mixtures for long periods. Three studies in particular were well-conducted and well-reported. Kimbrough et al. (1975) exposed female rats to 0 and 100 ppm Aroclor 1260 in the diet for 20-21 months and the treated animals had a high frequency of liver tumors. The National Cancer Institute (NCI 1978) reported significant dose-related increases of liver tumors and hyperplastic nodules in the livers of rats fed diets containing 0, 25, 50, or 100 ppm Aroclor 1254 for 104-105 weeks. Schaeffer et al. (1984) reported on a comparative study in male rats of the carcinogenicity and chronic toxicity of Clophen A60 (equivalent to Aroclor 1260) and Clophen A30 (equivalent to Aroclor 1242) fed at concentrations of 100 ppm in the diets for up to 832 days. A strong carcinogenic effect of Clophen A60 (hepatocellular carcinomas and/or neoplastic nodules) and a weaker carcinogenic effect of Clophen A30 (neoplastic nodules) were reported.

Studies have suggested that PCB mixtures can act to promote or inhibit the action of other carcinogens in rats and mice. Preston et al. (1981) reported that exposure to Aroclor 1254 at a concentration of 100 ppm in the diet for 18 weeks markedly augmented the development of liver tumors initiated by diethylnitrosamine in rats. In two-stage bioassay systems in rat livers using diethylnitrosamine as an initiator, Clophen A50 (equivalent to Aroclor 1254) and Aroclor 1254 were shown to have promoting activities by Oesterle and Deml (1984) and Pereira et al. (1982), respectively. The Oesterle and Deml (1984) study is noteworthy as the only dose-response study of cancer promotion by PCBs.

PCB mixtures and several isomers have been studied for genotoxic effects, and the results have been generally negative (EPA 1985a). Studies of the

metabolic activation of PCBs and their ability to induce microsomal enzymes have demonstrated that PCB isomers and their metabolites do bind to macromolecules, including DNA, with varying degrees of affinity. This suggests that PCBs are capable of damaging DNA, but it is unclear whether the damage is repairable or likely to produce permanent alterations in the genome. Their ability to induce enzymes responsible for the metabolic activation of other known mutagens and carcinogens may be the greatest genetic hazard posed by PCBs.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

EPA Carcinogen Assessment Group (EPA 1985a) calculated a low-level cancer potency factor for PCBs based on a study of rats exposed to Aroclor 1260 (Kimbrough et al., 1975). The data on liver tumor incidence in the rat study was used in the linearized multistage model to calculate 95% upper confidence limits on risk. The risks determined using this approach are unlikely to underestimate the actual risks posed by exposure to low levels of PCBs in the environment and may overestimate risk. The carcinogenic potency factor for lifetime exposure to PCBs is 4.34 $(mg/kg/day)^{-1}$, indicating that a risk of 4.34 (upper bound) is associated with continuous lifetime exposure to a dose of 1 mg/kg/day. EPA (1984) classified the weight of the evidence for carcinogenicity as B2--Probable Human Carcinogen based on sufficient evidence in animal bioassays and inadequate evidence from studies in humans. Based on the Schaeffer et al. (1984) study, one would expect less chlorinated PCB mixtures to be less potent, but potency factors for mixtures other than Aroclor 1260 have not been calculated.

EPA (1980) derived an Ambient Water Quality Criterion (AWQC) for the protection of human health from the potential carcinogenic effects of PCBs through ingestion of contaminated water and contaminated aquatic organisms. The recommended AWQC corresponding to a 10^{-6} incremental increase of cancer risk over a lifetime is 0.079 ng/liter.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) recommends a time-weighted average Threshold Limit Value (TLV) of 1 mg/m^3

for Arochlor 1242, which should offer good protection against systemic intoxication. The ACGIH also recommends a time-weighted average TLV of 0.5 mg/m^3 for Arochlor 1254.

REFERENCES

AIBRO, P.W., and L. FISHBEIN. 1972. Intestinal absorption of polychorinated biphenyls in rat. Bull. Environ. Contam. Toxicol. 8:26

ALLEN, J.R., W.A. HARGRAVES, M.T. HSIA, et. al. 1979. Comparative toxicology of chlorinated compounds on mammalian species. Pharmacol. Ther. 7:513-547

ALLEN, J.R., and BARSOTTI, D.A. 1976. The effects of transplacental and mammary movement of PCBs on infant rhesus monkeys. Toxicology 6:331-340

ALLEN, J.R., BARSOTTI, D.A., and CARSTENS, L.A. 1980. Residual effects of polychlorinated biphenyls on adult nonhuman primates and their offspring. J. Toxicol. Environ. Health 6:55-66

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

BARSOTTI, D.A., F.J. MARLAR, and J.R. ALLEN. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet. Toxicol. 14:99

BOWMAN, R.E., and HEIRONIMUS, M.P. 1981. Hypoactivity in adolescent monkeys perinatally exposed to PCBs and hyperactive as juveniles. Neurobeh. Toxicol. Teratol. 3:15-18

BURSE,, V.W., KIMBROUGH, R.D., VILLANUEVA, E.C., JENNINGS, R.W., LINDER, R.E., and SOVOCOOL, G.W. 1974. Polychlorinated biphenyls--Storage, distribution, excretion and recovery: Liver morphology after prolonged dietary ingestion. Arch. Environ. Health 29:301-307

CHASE, K.H., WONG, O., THOMAS, D., BERNEY, B.W., and SIMON, R.K. 1982. Clinical and metabolic abnormalities associated with occupational exposure to polychlorinated biphenyls. J. Occup. Med. 24:109-114

DEICHMANN, W.B. 1981. Halogenated cyclic hydrocarbons. In Clayton, G.D., and Clayton, F.E., eds. Patty's Industrial Hygiene and Toxicology. 3rd ed. John Wiley and Sons, New York

EMMETT, E.A., AGNEW, J., BLEEKER, M.L., FERRARA, J.M., LEVIN, B.K., JEFFREYS, J., and MARONI, M. 1983. Health Effects of PCB Exposure of GSA Switchgear Employees. NIOSH Health Hazard Evaluation Program Medical Report, Technical Assistance Request No. 80-7. The Johns Hopkins University, Baltimore, Maryland

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Polychlorinated Biphenyls. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. EPA/440/5-80-068. NTIS PB 81-117798

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Polychlorinated Biphenyls. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-004

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Health Effects Criteria Document on Polychlorinated Biphenyls. Office of Drinking Water. Washington, Final Draft

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms. Fed. Reg. 50:47001-47002. (November 13, 1985)

FEIN, G.G., JACOBSON, J.L., JACOBSON, S.W., SCHWARTZ, P.M., and DOWLER, J.K. 1984a. Prenatal exposure to polychlorinated biphenyls: Effects on birth size and gestational age. J. Pediatr. 105:315-320

FEIN, G.G., JACOBSON, J.L., JACOBSON, S.W., and SCHWARTZ, P.M. 1984b. Intrauterine Exposure of Humans to PCBs: Newborn Effects NTIS BP84-188887, EPA 600/3-84-060

FISCHBEIN, A., M.S. WOLFF, R. LILIS, J. THORNTON, and I. J. SELIKOFF. 1979. Clinical finding among PCB-exposed capacitor manufacturing workers. Ann. NY Acad. Sci. 320:703

HODGE, H.C., and J.H. STERNER, 1949. Tabulation of toxicity classes. Am. Ind. Hyg. Assoc. Q 10:93

JACOBSON, J.L., FEIN, G.G., JACOBSON, S.W., SCHWARTZ, P.M,., and DOWLER, J.K. 1984. The transfer of polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs) across the human placenta and into maternal milk. Am. J. Public Health 74:378-379

JACOBSON, S.W., JACOBSON, J.L., SCHWARTZ, P.M., and FEIN, G.G. 1983. Intrauterine exposure of human newborns to PCBs: Measures of exposure. In D'Itri, F.M., and Kamrin, M.A., eds., PCBs: Human and Environmental Hazards. Butterworth Publishers, Boston. Pp. 311-343

KIMBROUGH, R.D., R.E. LINDER, and T.B. GAINES. 1972. Morphological changes in livers of rats fed polychlorinated biphenyls. Arch. Environ. Health 25:354

KIMBROUGH, R.D., R.A. SQUIRE, R.E. LINDER, J.D. STRANDBERG, R.J. MONTALI, and V.W. BURSE. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. J. Natl. Cancer Inst. 55:1453

KIMBROUGH, R. et al. 1978. Animal toxicity. Environ. Health Perspect. 24:173

KOLLER, L.D. 1977. Enhanced polychlorinated biphenyl lesions in Moloney leukemia virus-infected mice. Clin. Toxicol. 11:107-116

LINDER, R.E., T.B. GAINES, and R.D. KIMBROUGH. 1974. The effect of PCB on rat reproduction. Food Cosmet. Toxicol. 12:63

MARONI, M., COLOMBI, A., CANTONI, S., FERIOLI, E., and FOA, V. 1981. Occuptional exposure to polychlorinated biphenyls in electrical workers. I. Environmental and blood polychlorinated biphenyls concentrations. Br. J. Ind. Med. 38:55-60

MARONI, M., COLUMBI, A., ARBOSTI, G., CANTONI, S., and FOA, V. 1981. Occupational exposure to polychlorinated biphenyls in electrical workers. II. Health effects. Br. J. Ind. Med. 38:55-60

MATTHEWS, H.B. 1983. Metabolism of PCBs in Mammals: Routes of Entry, Storage and Excretion. In D'Itri, F.M., and Kamrin, M.A., eds. PCBs: Human and Environmental Hazards. Butterworth, Boston.

McNULTY, W.P., BECKER, G.M., and CORY, H.T. 1980. Chronic toxicity of 3,4,3',4- and 2,5,2',5'-tetrachlorobiphenyls in rhesus macaques. Toxicol. Appl. Pharmacol. 56:182-190

NATIONAL CANCER INSTITUTE (NCI). 1978. Bioassay of Aroclor 1254 for Possible Carcinogenicity. Cas. No. 27323-18-8. NCI Carcinogenesis Technical Report Series No. 38. DHEW Publication No. (NIH)78-838

NATIONAL RESEARCH COUNCIL OF CANADA (NRCC). 1978. Polychlorinated biphenyls: Biological criteria for an assessment of their effects on environmental quality. NRCC. No. 16077. Ottawa.

OESTERLE, D., and DEML, E. 1984. Dose-dependent promoting effect of polychlorinated biphenyls on enzyme-altered islands in livers of adult and weanling rats. Carcinogenesis 5:351-355

PEREIRA, M.A., S.L. HERREN, A.L. BRITT, and M.M. KHOURY. 1982. Promotion by polychlorinated biphenyls of enzyme altered foci in rat liver. Cancer Letters 15:185-190

PRESTON, B.D., J.P. VAN MILLER, R.W. MOORE, AND J.R. ALLEN. 1981. Promoting effects of polychlorinated biphenyls (Aroclor 1254) and polychlorinated dibenzofuran-free Aroclor 1254 on diethylnitrosamine-induced tumorigenesis in the rat. J. Natl. Canc. Inst. 66:509-515

RINGER, R.K. 1983. Toxicology of PCBs in Mink and Ferrets. In D'Itri, F.M., and Kamrin, M.A., eds. PCBs: Human and Environmental Hazards. Butterworth, Boston.

SAFE, S. 1980. Halogenated biphenyls terphenyls, naphthalenes, dibenzodioxins and related products. Metabolism, uptake, storage, and bioaccumulation. Toxicol. Environ. Health 4:81-107

SCHAEFFER, E., GREIM, H., and GOESSNER, W. 1984. Pathology of chronic polychlorinated biphenyl (PCB) feeding in rats. Toxicol. Appl. Pharmacol. 75:278-288

SLEIGHT, S.D. Pathologic effects of PCBs in mammals. In D'Itri, F.M., and Kamrin, M.A., eds. PCBs: Human and Environmental Hazards. Butterworth, Boston.

SMITH, A.B. et al. 1982. Metabolic and health consequences of occupational exposure to polychlorinated biphenyls. Br. J. Ind. Med. 39:361-369

THOMAS, P.T., and HINSDILL, R.D. 1978. Effect of polychlorinated biphenyls on the immune responses of rhesus monkeys and mice. Toxicol. Appl. Pharmacol. 44:41-51

TRUELOVE, J., GRANT, D., MES, J., TRYPHONAS, H., and ZAWIDZKA, Z. 1982. Polychlorinated biphenyl toxicity in the pregnant cynomolgus monkey: A pilot study. Arch. Environ. Contam. Toxicol. 11:583-588

WESTER, R.C., A.W. BUCKS, H.I. MAIBACK, and J. ANDERSON. 1983. Polychlorinated Biphenyls (PCBs): Dermal Absoprtion, Systemic Elimination, and Dermal Wash Efficiency. J. Toxicol Environ. Hlth. 12:511

POLYCYCLIC AROMATIC HYDROCARBONS

INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are a diverse class of compounds consisting of two or more fused aromatic rings. They are formed during the incomplete combustion of materials containing carbon and hydrogen and are ubiquitous in the modern environment. PAHs are commonly found as constituents of coal tar, soots, vehicular exhausts, cigarettte smoke, certain petroleum products, road tar, mineral oils, creosote, and many cooked foods.

A number of reviews have been prepared on the toxicology of the PAHs. The Environmental Protection Agency (EPA) prepared an Ambient Water Quality Criteria Document on the general class of polynuclear aromatic hydrocarbons (EPA 1980a) and also prepared Criteria Documents on several specific PAHs, including acenaphthene, fluoranthene, and naphthalene (EPA 1980b-d). More recently, EPA (1984a-f) prepared Health Effects Assessments for PAHs as a class, for coal tars, and for the individual compounds benzo(a)pyrene (B(a)P), naphthalene, phenanthrene, and pyrene. In addition to the EPA documents, Santodonato et al. (1981) prepared a review and risk assessment of polynuclear aromatic hydrocarbons, and the International Agency for Research on Cancer (IARC) reviewed the toxicity and carcinogenicity of a large number of individual PAHs and PAH-containing mixtures (IARC 1973, 1983, 1984, 1985).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Acute effects from direct contact with PAHs and related materials are limited primarily to phototoxicity. Phototoxicity is caused by exposure to a toxic substance followed by exposure to sunlight. The primary effects are dermatitis—skin reddening, itching, and burning. NIOSH (1977) reviewed the phototoxic effects of exposure to coal tar and found that phototoxicity can result from a single 90-minute exposure to a 1% solution of coal tar.

These dermatoses usually disappear when contact with the sensitizer is eliminated.

The polycyclic aromatic hydrocarbons have been shown to cause cytotoxicity in rapidly proliferating cells throughout the body, with the hematopoietic system, lymphoid system, and testes frequently noted as targets (Santodonato et al. 1981). This effect appears to be due to inhibition of DNA replication by the PAHs. Destruction of the sebaceous glands, hyperkeratosis, hyperplasia, and ulceration have been observed in mouse skin following dermal application of the carcinogenic PAHs, with the degree of induced morphological changes appearing to correlate with the carcinogenic activity. However, it does not seem that this dermal toxicity is necessary or sufficient for carcinogenesis (Santodonato et al. 1981). It should be noted that similar types of dermatitis have been observed in workers exposed to such PAH-containing materials as coal tar and mineral oil. The carcinogenic PAHs have also been shown to have an immunosuppressive effect in animals. Again, it is not clear what relationship, if any, this immunosuppression has with carcinogenesis.

Some of the noncarcinogenic PAHs have been shown to cause systemic toxicity, but these effects are generally seen only at rather high doses (Santodonato et al. 1981). For example, slight morphological changes in the livers and kidneys of rats have been reported following oral administration of acenaphthene. Oral administration of naphthalene to rabbits has resulted in cataract formation.

Nonneoplastic lesions are seen in animals exposed to the more potent carcinogenic PAHs only after exposure to levels well above those required to elicit a carcinogenic response. Consequently, carcinogenicity is the toxic effect of greatest public health concern following exposure to materials containing carcinogenic PAHs. A number of the PAHs have been shown to be potent carcinogens, producing tumors both at the site of application and systemically, in several different animal species, when administered by any of a number of routes. For example, Rigdon and Neal (1969) reported gastric tumors, pulmonary adenomas and leukemias in mice fed B(a)P, and

intratracheal instillation of a number of PAHs has been shown to cause lung tumors in both mice and hamsters (Santodonato et al. 1981). In addition, IARC (1984, 1985) have noted that occupational exposure to coal soot, coal tar and pitch, coal tar fumes, and some impure mineral oils causes cancer in humans at several sites, including the skin, and concluded that there is sufficient evidence that soots, tars, and some mineral oils are carcinogenic in humans. Fractionation procedures have demonstrated that the PAHs are the carcinogenic agents in coal tar.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Not all PAHs have been demonstrated to be carcinogenic in animals, and some carcinogenic PAHs are clearly more potent than others (IARC 1983). A number of factors have been shown to influence the relative carcinogenic potencies of the PAHs. These include planarity of the molecule, cellular absorption, binding affinity, the presence or absence of a particular molecular structure, and the electron configuration of the molecule (Dipple et al. 1984, Frierson et al. 1986). In addition, genetic differences in the exposed animals, particularly in their ability to produce aryl hydrocarbon hydroxylase (AHH), have been shown to influence carcinogenic potency. Finally, the PAHs are not ultimate carcinogens but must be metabolized before they become biologically activated. A complete description of the complex metabolism of these compounds is beyond the scope of this report, but a detailed review of the factors influencing the carcinogenicity of the PAHs and metabolism of these compounds can be found in Dipple et al. (1984), Santodonato et al. (1981), and Frierson et al. (1986). For purposes of risk assessment, it is sufficient to note that the potencies of different PAHs vary over a wide range and that a number of factors, including factors specific to the chemical, the host animal, and the circumstances of exposure, affect carcinogenic potency.

For practical purposes, the PAHs are often separated into two categories, the "carcinogenic" and the "noncarcinogenic" PAHs. This is a somewhat misleading categorization as many of the "noncarcinogenic" PAHs have been shown to have some, albeit weak, carcinogenic activity, or to act as promoters

or cocarcinogens. A more accurate designation would be to differentiate between potent carcinogens, weak carcinogens, and noncarcinogens. Another factor complicating the assessment of risks posed by the PAHs is that they do not occur alone in nature, but occur as complex mixtures containing numerous PAHs of varying carcinogenic potencies. The potential interactions of the individual PAHs present as components of these mixtures must be addressed in attempting to quantify the carcinogenic risks posed by exposure to the mixtures.

The approach adopted by EPA (1980a, 1984a) as the basis for risk assessment is to apply a carcinogenic potency factor calculated from assays on B(a)P to the subclass of carcinogenic PAHs within the mixture that is to be assessed. This approach involves three assumptions: (1) that all carcinogenic PAHs have the same potency as B(a)P; (2) that their effects are additive; and (3) that the noncarcinogenic PAHs do not contribute to the carcinogenic effects of the mixture. Although there is limited empirical evidence to support assumptions (2) and (3), assumption (1) may lead to large overestimates of risk because B(a)P is one of the most potent carcinogens among the PAHs and is usually present only in a small percentage of the total mixture.

EPA (1980a, 1984a) calculated a value of 11.5 $(mg/kg/day)^{-1}$ as the carcinogenic potency factor (upper bound on lifetime risk) for oral exposure to the carcinogenic PAHs, based on the study of Neal and Rigdon (1967) in which oral administration of B(a)P led to forestomach tumors in mice. EPA (1984a) calculated a cancer potency factor for inhalation of B(a)P based on the study of Thyssen et al. (1981 as cited in EPA 1984a). This assay evaluated the production of respiratory tract tumors in hamsters using B(a)P at concentrations of 2.2-9.5 mg/m^3. The linearized multistage model yielded a carcinogenic potency factor of 6.11 $(mg/kg/day)^{-1}$.

EPA's Carcinogen Assessment Group has not reported a risk assessment for dermal exposure to the carcinogenic PAHs. Santodonato et al. (1981) performed risk assessments for both dermal and oral exposure and indicated that

B(a)P was more potent when applied dermally than when administered orally. A number of factors may account for this difference in relative potency and a complete derivation of a dermal potency factor is beyond the scope of this profile.

In addition to quantification of the effects of individual PAHs, EPA developed a cancer potency factor for inhalation of coal tar pitch volatiles (EPA 1984b). This study evaluated epidemiological data from exposure of coke oven workers to between 0 and greater than 700 mg/m^3-month coal tar vapors. The equivalent incremental risk calculated from the study was 3.2 $(mg/kg/day)^{-1}$. Coal tar pitch volatiles were classified in Group A—Human Carcinogen.

IARC (1983) in reviewing the carcinogenicity of the PAHs, indicated those for which there was sufficient, limited, inadequate, or adequate negative evidence of carcinogenicity (Table 1). The more potent carcinogens are almost certainly included within the group for which sufficient evidence of carcinogenicity is available.

TABLE 1

CLASSIFICATION OF PAHs ACCORDING TO EVIDENCE FOR CARCINOGENICITY

Chemicals for which there is sufficient evidence that they are carcinogenic in animals:

Benzo(a)anthracene
Benzo(b)fluoranthene
Benzo(j)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Dibenzo(a,h)acridine
Dibenzo(a,j)acridine
Dibenzo(a,h)anthracene
7H-Dibenzo(c,g)carbazole
Dibenzo(a,e)pyrene
Dibenzo(a,h)pyrene
Dibenzo(a,i)pyrene
Dibenzo(a,l)pyrene
Indeno(1,2,3-c,d)pyrene
5-Methylchrysene

Chemicals for which there is limited evidence that they are carcinogenic in animals:

Anthranthrene
Benzo(c)acridine
Carbazole
Chrysene
Cyclopenta(c,d)pyrene
Dibenzo(a,c)anthracene
Dibenzo(a,j)anthracene
Dibenzo(a,e)fluoranthene
2-, 3-, 4-, and 6-Methylchrysene
2- and 3-Methylfluoranthene

Chemicals for which the evidence is inadequate to assess their carcinogenicity:

Benzo(a)acridine
Benzo(g,h,i)fluoranthene
Benzo(a)fluorene
Benzo(b)fluorene
Benzo(c)fluorene
Benzo(g,h,i)perylene
Benzo(c)phenanthrene
Benzo(e)pyrene
Coronene
1,4-Dimethylphenanthrene
Fluorene
1-Methylchrysene
1-Methylphenanthrene
Perylene
Phenanthrene
Triphenylene

Chemicals for which the available data provide no evidence that they are carcinogenic:

Anthracene
Fluoranthene
Pyrene

SOURCE: IARC 1983

REFERENCES

DIPPLE, A., MOSCHEL, R.C., and BIGGER, C.A.H. 1984. Polynuclear aromatic carcinogens. In Searle, C.E. ed. Chemical Carcinogens-Second edition. ACS Monograph 182. American Chemical Society. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980a. Ambient Water Quality Criteria for Polynuclear Aromatic Hydrocarbons. Office of Water Regulations and Standards, Washington D.C. October 1980. EPA 440/5-80-069

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980b. Ambient Water Quality Criteria for Acenaphthene. Office of Water Regulations and Standards, Washington, D.C. October 1980. EPA 440/5-80-015

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980c. Ambient Water Quality Criteria Document for Fluoranthene. Office of Water Regulations and Standards. Washington, D.C. October 1980. EPA 440/5-80-049

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980d. Ambient Water Quality Criteria for Naphthalene. Office of Water Regulations and Standards. Washington, D.C. October 1980. EPA 440/5-80-059

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons (PAH). Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-013

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Health Effects Assessment for Coal Tars. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-024

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984c. Health Effects Assessment for Benzo(a)pyrene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-022

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984d. Health Effects Assessment for Naphthalene. Environmental Criteria and Assessment Office, Cincinatti, Ohio. September 1984. EPA 540/1-86-014

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984e. Health Effects Assessment for Phenanthrene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-029

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984f. Health Effects Assessment for Pyrene. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-030

FRIERSON, M.R., KLOPMAN, G., and ROSENKRANZ, H.S. 1986. Structure-activity relationships (SARs) among mutagens and carcinogens: A Review. Environ. Mutagen. 8:283-327

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1973. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Volume 3. Certain Polycyclic Aromatic Hydrocarbons and Heterocyclic Compounds. World Health Organization, Lyon, France

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1983. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Vol. 32: Polynuclear Aromatic Compounds; Part 1, Chemical, Environmental, and Experimental Data. World Health Organization, Lyon, France

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1984. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 33: Polynuclear Aromatic Compounds, Part 2, Carbon Blacks, Mineral Oils and Some Nitroarenes. World Health Organization, Lyon, France

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1985. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 35: Polynuclear Aromatic Compounds, Part 4, Bitumens, Coal-tar and Derived Products, Shale-oils, and Soots. World Health Organization, Lyon, France

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977. Criteria for a Recommended Standard-Occupational Exposure to Coal Tar Products. Washington, D.C. DHEW (NIOSH) 78-107

NEAL, J. and RIGDON, R.H. 1967. Gastric tumors in mice fed benzo(a)pyrene: A quantitative study. Tex. Rep. Biol. Med. 25:553-557

RIGDON, R., and NEAL, J. 1969. Relationship of leukemia to lung and stomach tumors in mice fed benzo(a)pyrene. Proc. Soc. Exp. Biol. Med. 130:146-148

SANTODONATO, J., HOWARD, P., and BASU, D. 1981. Health and ecological assessment of polynuclear aromatic hydrocarbons. J. Environ. Pathol. Toxicol. 5:1-364

THYSSEN, J., ALTHOFF, J., KIMMERLE, G., and MOHR, U. 1981. Inhalation studies with benzo(a)pyrene in Syrian golden hamsters. J. Natl. Cancer Inst. 66:575-577

TOLUENE

INTRODUCTION

Toluene occurs naturally as a component of petroleum oil. It is used in the production of benzene and other organic solvents and as a solvent for paints, coatings, gums, oils, and resins.

PHARMACOKINETICS

In humans, toluene is absorbed quickly through the respiratory tract; dermal absorption of aqueous toluene is directly related to concentration (EPA 1985a). Because of its lipophilic nature and low water solubility, toluene is expected to distribute and accumulate in lipid tissue (EPA 1985b). In humans and animals, toluene is excreted via the urine as the metabolite hippuric acid and through pulmonary exhalation as the unchanged compound (EPA 1985a).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENICITY

Toluene has not been adequately tested for carcinogenicity. A chronic (106-week) oral bioassay of toluene conducted in F344 rats of both sexes resulted in no carcinogenic effects (CIIT 1980 as cited in EPA 1985b). Gross and microscopic examination of tissues and organs revealed no increase in neoplastic tissues or tumor masses among rats treated at 30, 100, or 300 ppm when compared with controls. However, the highest dose used did not approach the maximum tolerated dose (MTD); therefore, it has been suggested that toluene may not have been adequately tested for carcinogenicity. The National Toxicology Program is currently conducting an inhalation carcinogenicity bioassay in rats and mice (NTP 1985 as cited in EPA 1985a).

MUTAGENICITY

Toluene has been tested for genotoxicity by many investigators using various assay methods and has not been demonstrated to be genotoxic (EPA 1985a).

TERATOGENICITY/REPRODUCTIVE EFFECTS

Toluene administered by gavage to CD-1 mice at 260 mg/kg/day on days 6-15 of gestation caused embryo deaths, and higher doses (430 or 870 mg/kg/day) also caused fetal and maternal toxicities and cleft palates (Nawrot and Staples, 1979 Abstract as cited in EPA 1985b).

ACUTE/CHRONIC EFFECTS

The oral LD_{50} in rats is between 5,300 and 7,500 mg/kg (EPA 1985b). The LC_{50} for inhaled toluene is 4,618 ppm after a 6-hour exposure in rats (EPA 1985a). The dermal LD_{50} in rabbits is 12.2 g/kg (EPA 1985a). In experimental animals, acute oral and inhalation exposures to toluene result in CNS depression and histological changes in the lungs, liver, and kidneys (EPA 1985b). In humans, the major acute effects of toluene (375-1,500 mg/m^3) are CNS depression and narcosis (EPA 1985b).

Toxic effects following prolonged exposure of experimental animals to toluene are similar to those seen following acute exposure, predominantly to the CNS, liver, kidneys, and lungs (EPA 1985b). In humans, chronic exposures to toluene vapors at levels of approximately 200 and 800 ppm have been associated with CNS and, possibly, peripheral nervous system effects, hepatomegaly, and hepatic and renal function changes (EPA 1985b).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying EPA's criteria for evaluating the overall weight of evidence of carcinogenicity to humans (EPA 1986a), toluene is most appropriately classified in Group D--Not Classified. This category indicates that the evidence for carcinogenicity in animals is inadequate (EPA 1986a).

The ADI Work Group of the Risk Assessment Forum (EPA 1986b) has identified an oral risk reference dose (RfD) of 0.3 mg/kg/day (20 mg/day for a 70-kg man). The risk RfD is based on a 24-month chronic toxicity study conducted in male and female F344 rats (CIIT 1980). Toluene was administered by inhalation at 30, 100, or 300 ppm to 120 male and female F344 rats, 6 hours/day, 5 days/week. The same number of animals was used as a control. Clinical chemistry, hematology, and urinalysis testing were conducted at 18 and 24 months. All parameters measured at the termination of the study were normal except for a dose-related reduction in hematocrit values in females exposed at 100 and 300 ppm toluene. Based on these findings, a no-observed-adverse-effect level (NOAEL) of 300 ppm was identified and an oral RfD of 0.3 mg/kg/day was derived.

The EPA Office of Drinking Water developed one-day, ten-day, and lifetime health advisories (HAs) for toluene (EPA 1985a). The one-day HA values, derived for a 10-kg child and a 70-kg adult, are 18.0 and 63.0 mg/liter, respectively. The ten-day HA values are based on the accumulated data on human inhalation exposure to toluene which indicate that approximately 100 ppm toluene for up to 8 hours/day causes no apparent adverse effects in humans. The ten-day HA values, derived for a 10-kg child and a 70-kg adult, are 6.0 and 21.0 mg/liter, respectively. The lifetime health advisory was based on the study by CIIT (1980) described above. A NOAEL of 300 ppm was identified and used to derive the risk reference dose of 0.288 mg/kg/day. The resultant adjusted acceptable daily intake (AADI) for toluene was 10.1 mg/liter. EPA's Office of Drinking Water has proposed a recommended maximum contaminant level (RMCL) of 2 mg/liter for toluene based on consideration of a source contribution factor of 20%.

The American Conference of Governmental Industrial Hygienists has recommended a time-weighted average threshold limit value of 100 ppm (approximately 435 mg/m^3) for occupational exposure to toluene.

SUMMARY OF TOLUENE CRITERIA

EPA carcinogen classification	Group D
One-day HA:	
Child	18.0 mg/liter
Adult	63.0 mg/liter
Ten-day HA:	
Child	6.0 mg/liter
Adult	21.0 mg/liter
Lifetime HA (AADI)	10.1 mg/liter
Oral RfD	0.3 mg/kg/day

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY (CIIT). 1980. A twenty-four month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. Executive Summary and Data Tables. October 15, 1980

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Toluene. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-033

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Draft Health Advisory for Toluene. Office of Drinking Water, Washington, D.C. September 30, 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Drinking Water Criteria Document for Toluene. Office of Drinking Water, Washington, D.C. March 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Guidelines for carcinogenic risk assessment. Fed. Reg. 51:33992-34003 (September 24, 1986)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Verified Reference Doses (RFDs) of the EPA. Office of Health and Environmental Assessment, Washington, D.C. January 1986. ECAO-CIN-475

NAWROT, P.S., and STAPLES, R.E. 1979. Embryo-fetal toxicity and teratogenicity of benzene and toluene in the mouse. Teratology 19:41A (Abstract)

TRICHLOROETHYLENE

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Trichloroethylene is a central nervous system depressant from acute and chronic exposure. High level exposure can result in death due to respiratory and cardiac failure. Trichloroethylene was once used as a general anesthetic, but its use was discontinued due to longer-term CNS effects. The longer-term effects may have been due to impurities introduced by soda lime used to remove carbon dioxide (EPA 1980).

The extent of trichloroethylene absorption after oral ingestion is virtually complete. With air exposure, absorption is proportional to concentration and duration of exposure. Trichloroethylene is eliminated by pulmonary excretion of the unchanged parent compound and by liver metabolism of urinary metabolites (EPA 1985a).

The hepatotoxic potential of trichloroethylene has been evaluated in human and animal studies. Animal studies have revealed transient increased liver weights, but relative liver weights decreased post-exposure (Kjellstrand et al. 1983 as cited in EPA 1985a). Observations of liver or renal dysfunction in workers have been infrequent, and factors other than trichloroethylene probably were more causally related to the hepatorenal disturbances noted (EPA 1985a).

Industrial use of trichloroethylene is often associated with dermatological problems. These include reddening and skin burns on contact, and dermatitis resulting from vapors. These effects are usually the result of contact with concentrated solvent, however, and no effects have been reported with exposure to trichloroethylene in dilute, aqueous solutions (EPA 1985a).

Studies investigating the carcinogenic potential of trichloroethylene have been carried out, and two of these studies revealed significant increases

in the incidence of liver tumors among both sexes of B6C3F1 mice exposed by gavage (NCI 1976, NTP 1982 as cited in EPA 1985a).

There is disagreement in the scientific community about the relevance of mouse liver tumors as indicators of human cancer risk. Several strains of laboratory mice, including the B6C3F1 hybrid, appear to develop a high and variable proportion of liver tumors with or without exposure to chemicals. Certain scientists believe that the increased incidence of mouse liver tumors should be treated in the same manner as the increased incidence of tumors at other rodent organ sites, while others believe that mouse liver tumors are an experimental artifact which is not relevant to human hazard (EPA 1985b).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

EPA's Science Advisory Board examined the toxicology of trichloroethylene and its ranking under the IARC criteria. The majority of the Committee members felt that the compound should be classified in IARC Category 3 (compound cannot be classified as to its carcingenicity in humans), while one member felt that IARC category 2B (probable human carcinogen) was more appropriate. The committee concluded that a definitive scientific opinion concerning the carcinogenicity of trichloroethylene could not be made at that time because the interpretation of male mouse hepatocellular carcinomas is uncertain and the animal evidence is limited (EPA 1985b).

EPA's Risk Assessment Forum classified trichloroethylene in Group B2 (probable human carcinogen, sufficient animal evidence of carcinogenicity and inadequate human evidence). The National Academy of Sciences has classified it as an animal carcinogen (EPA 1985b). Expressed in terms of relative potency, trichloroethylene ranks in the lowest quartile among the suspect or known human carcinogens evaluated by EPA's Carcinogen Assessment Group (CAG) (EPA 1985a).

CAG derived carcinogenic potencies (q_1^*) of 1.1×10^{-2} $(mg/kg/day)^{-1}$ for oral exposure and 4.6×10^{-3} $(mg/kg/day)^{-1}$ for inhalation exposure, derived

from the mouse liver tumor data in the NCI (1976) and NTP (1982) gavage studies (EPA 1984a).

EPA (1984b) determined a lifetime health advisory based on a study by Kimmerle and Eben (1973) that reported increased liver weights when rats were administered 55 ppm trichloroethylene for 14 weeks. The estimated excess cancer risk associated with lifetime exposure to drinking water containing 260 ug/liter of trichloroethylene is 8.2×10^{-5}.

STANDARDS AND CRITERIA

EPA carcinogen classification	Group B2
Oral carcinogenic potency (q_1^*)	1.1×10^{-2} $(mg/kg/day)^{-1}$
Inhalation carcinogenic potency (q_1^*)	4.6×10^{-3} $(mg/kg/day)^{-1}$
Proposed MCL (EPA 1985c)	5 ug/liter
Lifetime Health Advisory	260 ug/liter (for noncarcinogenic effects only)

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Trichloroethylene. Office of Water Planning and Standards. Washington, D.C. EPA 440/5-80-077

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Effects Assessment for Trichloroethylene. Environmental Criteria and Assessment Office. Cincinnati, Ohio. EPA 540/1-86-046

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Draft Health Advisory for Trichloroethylene. Office of Drinking Water. Washington, D.C. September 30. Draft

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Health Assessment Document for Trichloroethylene. Environmental Criteria and Assessment Office. Research Triangle Park, N.C. EPA/600/8-82/006F

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulations; volatile synthetic organic chemicals, final rule. Fed. Reg. 50:46880-46901 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985c. National primary drinking water regulations; volatile synthetic organic chemicals, proposed rule. Fed. Reg. 50:46902- 46933 (November 13, 1985)

KIMMERLE, G., and EBEN, A. 1973. Metabolism, excretion and toxicology of trichloroethylene after inhalation. I. Experimental exposure on rats. Arch. Toxicol. 30:115

KJELLSTRAND, P., HOLMQUIST, B., ALM, P., KANJE, M., ROMARE, S., JONSSON, I., MANNSON, L., and BJERKEMO. M. 1983. Trichloroethylene: Further studies of the effects on body and organ weights and plasma butyl cholinesterase activity in mice. Acta. Pharmacol. Toxicol. 53:375-384

NATIONAL CANCER INSTITUTE (NCI). 1976. Carcinogenesis bioassay of trichloroethylene. CAS No. 79-01-6. NCI-CG-TR-2.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1982. Carcinogenesis bioassay of trichloroethylene. CAS No. 79-01-6. NTP 81-84. NIH Publication No. 82-1799. Draft

VINYL CHLORIDE

Vinyl chloride and polyvinyl chloride (PVC) are synthetic chemicals used as starting materials in the rubber, paper, glass, and automotive industries. They are used in the manufacture of a wide variety of products, including electrical wire insulation, piping, food packaging materials, medical supplies, and building and construction products (EPA 1987a).

TOXICOKINETICS

Vinyl chloride is rapidly absorbed in rats following ingestion and inhalation exposure. Dermal absorption of vinyl chloride is minor. Absorbed vinyl chloride is distributed primarily to the liver and kidney, with lower levels found in muscle, lung, fat, spleen, and brain . The toxicity of vinyl chloride appears to be attributable to its metabolism in the liver to reactive polar metabolites. The metabolism to toxic metabolites is saturable between 105 and 220 ppm. At low doses of vinyl chloride, metabolites are excreted primarily in the urine. At high doses most of the solvent is expired as unchanged vinyl chloride (EPA 1980, 1985a,b).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENICITY

Occuptional exposure to vinyl chloride has been associated with an increased incidence of hepatic angiosarcoma. Vinyl chloride exposure has also been implicated in brain, lung, and hemolymphopoietic cancers in humans. Animal studies in several species support the findings of epidemiological studies. Chronic inhalation and ingestion of vinyl chloride has induced cancer in the liver (liver angiosarcomas and hepatocellular carcinomas) and in other tissues in rats and mice (IARC 1979).

MUTAGENICITY

The mutagenic effects of vinyl chloride have been demonstrated in metabolically activated systems using S. typhimurium, E. coli, yeast, germ cells of Drosophila, and Chinese hamster V79 cells. Vinyl chloride was effective in producing chromosome damage in rat bone marrow after a multiple-exposure regime. Tests for chromosomal aberrations in humans have yielded inconsistent results (EPA 1985a).

TERATOGENICITY/REPRODUCTIVE EFFECTS

Inhalation exposures of rats, rabbits, and mice to vinyl chloride did not induce teratogenic effects (EPA 1985a). Evidence for an association between human exposure to vinyl chloride and birth defects or fetal loss is conflicting (EPA 1987a). Potential effects on reproductive capacity have not been studied.

ACUTE/CHRONIC EFFECTS

At high inhalation exposure levels, workers have experienced dizziness, headaches, euphoria, and narcosis. In experimental animals, inhalation exposure to high levels of vinyl chloride can induce narcosis and death. Lower doses result in ataxia, narcosis, congestion, and edema of the lungs and hyperemia in the liver (EPA 1985a).

Chronic inhalation exposure of workers to vinyl chloride is associated with hepatotoxicity, central nervous system disturbances, pulmonary insufficiency, cardiovascular toxicity, gastrointestinal toxicity, and acro-osteolysis (EPA 1985a). Chronic studies of experimental animals exposed to vinyl chloride by inhalation or ingestion report effects involving the liver, spleen, kidneys, hematopoietic system, and skeletal system (EPA 1984).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Applying EPA's criteria for evaluating the overall weight of evidence of carcinogenicity to humans, vinyl chloride has been classified in Group A--Human Carcinogen (EPA 1984).

EPA (1984) reported carcinogenic potency factors (q_1^*) for exposure by inhalation and ingestion to vinyl chloride. The q_1^* for inhalation is based on an inhalation bioassay in rats (Maltoni and Lefemine 1975). Groups of 64 to 96 Sprague-Dawley rats were exposed to various concentrations of vinyl chloride for 4 hours a day, 5 days a week, for 52 weeks, and the survivors were sacrificed after 135 weeks. Angiosarcomas, particularly of the liver, were the predominant tumors observed. The linearized multistage model was fitted to the incidences of male and female rats with any type of malignant tumor (6/58, 10/59, 16/69, 22/59, and 32/59 in the 0-, 50-, 250-, 500-, and 2,500 ppm dose groups, respectively. The 6,000- and 10,000-ppm groups were not included in the final fitted model because the tumor incidence was said to have effectively plateaued at 51.7% and 62.3%. Using the linear nonthreshold model, the data of Maltoni and Lefemine (1975), and interspecies scaling factors, a human q_1^* of 2.5×10^{-2} $(mg/kg/day)^{-1}$ was calculated.

The q_1^* for oral exposure to vinyl chloride is based on a long-term ingestion study in rats (Feron et al. 1981). Groups of male and female Wistar rats were exposed to vinyl chloride via ingestion of polyvinyl chloride powder containing some unreacted monomer. The doses of vinyl chloride administered were 0, 1.7, 5.0, and 14.1 mg/kg/day. Dosing was continued for lifetimes with terminal sacrifices at 135 weeks for males and at 144 weeks for females. A significant dose-related increase in the incidence of hepatocellular carcinomas and hepatic angiosarcomas was observed in both males and females, with angiosarcomas becoming more prevalent with increasing doses. The linearized multistage model was fitted to the incidences of total female rats with tumors (2/57, 26/58, 42/59, in the 0-, 1.7-, 5.0-, and 14.1-mg/kg/day dose groups, respectively). The incidence of hepatocellular carcinoma was not included in these tallies; it was assumed that rats having hepatocellular carcinoma also had hepatic neoplastic nodules which were included in the tallies. In addition, the total number of animals bearing tumors in the high

dose group was arbitrarily reduced to one less than the total number of animals examined, so the data would fit the linear nonthreshold model used for estimation of carcinogenic potency. Using the data of Feron et al. (1981) and interspecies scaling factors, a human q_1^* of 2.3 $(mg/kg/day)^{-1}$ was calculated. The concentration in drinking water corresponding to a 10^{-6} excess lifetime cancer risk is 0.015 ug/liter. The EPA Carcinogen Assessment Group (CAG) is presently reassessing the cancer risk estimate based on the Feron et al. (1981) study by evaluating the more recent data by Til et al. (1983) which is an extension of the earlier Feron et al. (1981) work, but includes lower doses.

EPA promulgated a drinking water maximum contaminant level goal (MCLG) of zero because vinyl chloride is a human carcinogen. A drinking water MCL of 0.002 mg/liter has also been promulgated (EPA 1987b).

SUMMARY OF VINYL CHLORIDE CRITERIA

EPA carcinogen classification	Group A
Oral carcinogenic potency factor (q_1^*)	2.3 $(mg/kg/day)^{-1}$
Inhalation carcinogenic potency factor (q_1^*)	2.5×10^{-2} $(mg/kg/day)^{-1}$
MCLG	0 µg/liter
Final MCL	2 µg/liter
AWQC (10^{-6} upper-bound excess lifetime cancer risk)	0 (2 µg/liter)

REFERENCES

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Vinyl Chloride. Office of Water Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-078

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Vinyl Chloride. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-036

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document on Vinyl Chloride. Office of Drinking Water. Washington, D.C. January 1985. Final Draft

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. National primary drinking water regulations; volatile synthetic organic chemicals, final rule. Fed. Reg. 50:46880-46901 (November 13, 1985)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Health Advisory for Vinyl Chloride. Office of Drinking Water, Criteria and Standards Division. Washington, D.C. March 31, 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. National primary drinking water regulations—synthetic organic chemicals; monitoring for unregulated contaminants; final rule. Fed. Reg. 52:25690-25717 (July 8, 1987)

FERON, V.J,. HENDRIKSEN, C.F.M., SPEEK, A.J., TIL, H.P., and SPIT, B.J. 1981. Lifespan oral toxicity study of vinyl chloride in rats. Food Cosmet. Toxicol. 19:317-333

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979. IARC Monographs on the Evaluation of the Carcinogenic Risks of Chemicals to Humans. Vol. 20: Some Halogenated Hydrocarbons. World Health Organization, Lyon, France

MALTONI, C., and LEFEMINE, G. 1975. Carcinogenicity bioassays vinyl chloride. Current results. Ann. NY Acad. Sci. 246:195-218

TIL, H.P., IMMEL, H.R., and FERON, V.J. 1983. Lifespan Oral Carcinogenicity Study of Vinyl Chloride in Rats. Final Report. Civo Institutes TNO Report No. V 83 285/29109

STANDARDS AND CRITERIA

EPA carcinogen classification	Group D
ADI	0.01 mg/kg/day
Proposed RMCL	70 ug/liter

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of Threshold Limit Values and Biological Exposure Indices. 5th Ed. Cincinnati, Ohio

CERNA, M., and KYPENOVA, H. 1977. Mutagenic activity of chloroethylenes analysed by screening system tests. Mutat. Res. 46:214-215

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980. Ambient Water Quality Criteria for Dichloroethylenes. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-041

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Drinking Water Criteria Document for Dichloroethylenes. Office of Drinking Water, Washington, D.C. December 1984. PB 86-117785

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Proposed guidelines for carcinogen risk assessment; request for comments. Fed. Reg. 49:46294-46301. (November 23, 1984)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms. Fed. Reg. 50:46937-47025 (November 13, 1985)

FREUNDT, K.J., LIEBALDT, G.P., and LIEBERWIRTH, E. 1977. Toxicity studies on trans-1,2-dichloroethylene. Toxicology 7:141-153

GALLI, A., BAUER, C., BRONZETTI, G., CORSI, C., DEL CARRATORE, R., NIERI, R., and PAOLINI, M. 1982. [Genetic activity of 1,2-dichloroethylene. A. In vitro studies.] Boll. Soc. Ital. Biol. Sper. 58:860-863 (Italian) (Abstract from NLM Toxline Database)

GREIM, H. BONSE, G., RADWAN, Z., REICHERT, D., and HENSCHLER, D. 1975. Mutagenicity in vitro and potential carcinogenicity of chlorinated ethylenes as a function of metabolic oxiran formation. Biochem. Pharmacol. 24:2013-2017 (As cited in EPA 1980)

QUAST, J.F., HUMISTON, C.G., WADE, C.E., BALLARD, J., BEYER, J.E., SCHWETZ, R.W., and NORRIS, J.M. 1983. A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidene chloride. Fund. Appl. Toxicol. 3:55-62

XYLENES

INTRODUCTION

Xylenes are used as solvents for paints, inks, and adhesives and as components of detergents and other industrial and household products. Xylene has three isomers, o, m-, and p-xylene. These three compounds generally have similar chemical and biological characteristics and therefore will be discussed together.

PHARMACOKINETICS

Although the available data are limited, inference from metabolism and excretion studies suggests that absorption of orally administered xylene is nearly complete. Data from animals and humans suggest that approximately 60% of an inhaled dose is absorbed. Dermal absorption is reported to be minor following exposure to xylene vapor but may be significant following contact with the liquid (EPA 1985a). Elimination of xylenes is through urinary excretion of metabolites and through pulmonary exhalation of unchanged solvent (EPA 1985b).

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

CARCINOGENICITY

The National Toxicology Program (NTP) has tested xylene for carcinogenicity by administering the compound orally to rats and mice. Although the results have not been finalized, xylene does not appear to be carcinogenic in rats (EPA 1985b). Results have not been reported for mice.

MUTAGENICITY

Xylene was not found to be mutagenic in a battery of short-term tests (Litton Bionetics 1978).

TERATOGENICITY/REPRODUCTIVE EFFECTS

Xylene appears to be fetotoxic and may increase malformations in the offspring of exposed experimental animals. Except for one oral study in mice in which the incidence of cleft palates was increased, the available teratogenic studies have reported generally retarded skeletal development and body weight gains in fetuses (EPA 1985a).

ACUTE/CHRONIC EFFECTS

Most of the available toxicity data for xylene assesses the adverse effects associated with exposure by inhalation. Acute exposure to relatively high concentrations of xylene adversely affects the central nervous system and lungs, and can irritate the mucous membranes. The liver is reportedly affected by longer-term exposure to lower levels of xylene (EPA 1984, 1985a).

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

Using the criteria for evaluating the overall weight-of-evidence of carcinogenicity to humans proposed by EPA's Carcinogen Assessment Group (EPA 1986), xylene is most appropriately assigned to Group D--Not Classified because of inadequate data from animal studies.

The EPA Office of Drinking Water proposed a recommended maximum contaminant level (RMCL) of 0.44 mg/liter based on an inhalation study in rats, guinea pigs, monkeys, and dogs (Jenkins et al. 1970). The experimental animals were exposed continuously for 90 days at a dose level of 337 mg/m^3. No significant effects were observed with respect to body weight, hematology, and histopathological examination of treated animals. Using 337 mg/m^3 as the no-observed-adverse-effect level (NOAEL), an uncertainty factor of 1,000 for an animal study with few animals per dose level, and assuming consumption of 2 liters of water per day, a provisional adjusted acceptable daily intake of 2.2 mg/liter was calculated. The proposed RMCL was derived from this value by assuming a 20% drinking water contribution to total exposure (EPA 1985a,c).

EPA's Office of Drinking Water also drafted short-term assessments for xylene. One-day and longer-term health advisories of 42 mg/liter and 27.3 mg/liter were recommended. The one-day value was based on a NOAEL identified in a single-exposure inhalation study in humans, and the longer-term value was based on a NOAEL identified in a subchronic inhalation study in experimental animals (EPA 1985a).

The EPA (1984) health effects assessment for xylene derived oral subchronic acceptable intake (AIS) and chronic acceptable intake (AIC) values of 0.1 mg/kg/day and 0.01 mg/kg/day, respectively, based on a subchronic dietary exposure study in rats (Bowers et al. 1982). Groups of five male Long-Evans rats received approximately 10 mg/kg/day o-xylene in the diet for 1, 2, 3, or 6 months. No gross or light microscopic effects were observed. However, ultrastructural changes in liver morphology, which the EPA did not consider to be toxicologically significant, were noted. The exposure dose of 10 mg/kg/day was thus identified as the NOAEL and, incorporating an uncertainty factor of 100, the AIS for xylene was calculated. The AIC was derived by incorporating an additional uncertainty factor of 10. Based on available information, EPA assumed that until more experimental data become available it may be appropriate to apply the AIS and AIC derived from the o-xylene study to mixed xylenes and m-xylene, but not to p-xylene.

EPA (1984) derived an inhalation AIS based on a study in female Charles River CD rats exposed to mixed xylenes at 433 or 1,733 mg/m^3 on days 6 through 15 of gestation, 6 hours/day (Litton Bionetics 1978 as reported in EPA 1984). An increased number of fetuses, but not litters, with skeletal abnormalities was observed in the high dose group. The lower exposure concentration clearly defined a no-observed-effect level (NOEL) of 69 mg/kg/day. The NOEL was divided by an uncertainty factor of 100 to give an AIS of 0.69 mg/kg/day. Carpenter et al. (1975) defined an inhalation NOEL of 398 mg/kg for mixed xylenes in a subchronic inhalation study in rats. Applying an uncerainty factor of 1,000, EPA recommended an inhalation AIC of 0.398 mg/kg/day.

The American Conference of Governmental Industrial Hygienists (ACGIH 1986) has recommended a time-weighted average threshold limit value (TLV) of 100 ppm (approximately 435 mg/m^3) for occupational exposure to xylene. It is believed that at this concentration, irritant effects will be minimal.

SUMMARY OF XYLENE CRITERIA

EPA carcinogen classification	Group D
One-day HA	42 mg/liter
Longer-term HA	27.3 mg/liter
EPA Proposed RMCL	0.44 mg/liter
EPA Health Effects Assessment	
Oral AIS	0.1 mg/kg/day
Oral AIC	0.01 mg/kg/day
Inhalation AIS	0.69 mg/kg/day
Inhalation AIC	0.398 mg/kg/day

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio

BOWERS, D.E., CANNON, M.S., and JONES, D.H. 1982. Ultrastructural changes in livers of young and aging rats exposed to methylated benzenes. Am. J. Vet. Res. 43:679-683

CARPENTER, C.P., KINKEAD, E.R., GEARY, D.J., JR., SULLIVAN, L.J., and KING, J.M. 1975. Petroleum hydrocarbon toxicity studies. V. Animal and human response to vapors of mixed xylenes. Toxicol. Appl. Pharmacol. 33:543:558

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assesment for Xylene. Environmental Criteria and Asessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-066

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document for Xylenes (Final Draft). Environmental Criteria and Assessment Office, Cincinnati, Ohio. March 1985. ECAO-CIN-416. EPA 600/X-84-185-1

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Draft Advisory for Xylene. Office of Drinking Water, Washington, D.C. September

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985c. National primary drinking water regulations; synthetic organic chemicals, inorganic chemicals and microorganisms, proposed rule. Fed. Reg. 50:46936-47025. (November 13)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986. Guidelines for carcinogenic risk assessment. Fed. Reg. 51:33992-34003. (September 24)

JENKINS, L.J., JR., JONES, R.A., and SIEGAL, J. 1970. Long-term inhalation screening studies on benzene, toluene, o-xylene, and cumene on experimental animals. Toxicol. Appl. Pharmacol. 16:818-823

LITTON BIONETICS. 1978. Teratology studies in rats: Xylene. Final report to American Petroleum Institute, Washington, D.C. LBI Project No. 20698-5 (As cited in EPA 1984)

ZINC

INTRODUCTION

Zinc (Zn) is a silvery metal of low to intermediate hardness, atomic number 30 and atomic weight 65.38 g/mole. Primary uses of zinc in industry are as a component of dry cells and other batteries, in electrogalvanizing, and as alloys. Zinc compounds are used therapeutically as topical astringents, antidandruff products, antiseptics, and emetics.

QUALITATIVE DESCRIPTION OF HEALTH EFFECTS

Zinc is an essential element, and it is present in a number of metalloenzymes, including carbonic anhydrase carboxypeptidase, alcohol dehydrogenase, glutamic dehydrogenase, lactic dehydrogenase, and alkaline phosphatase (Vallee 1959 as cited by Hammond and Beliles 1980). Zinc is necessary for normal growth and development. Human dwarfism and a lack of sexual development have been related to Zn deficiency (Halsted et al. 1974 as cited by Hammond and Beliles 1980).

There is no evidence that zinc is carcinogenic. Studies which have been performed on zinc-containing organics failed to demonstrate teratogenic effects (Stokinger 1981).

Zinc salts of strong acids are astringent and corrosive. Upon ingestion they act as emetics, and they can cause symptoms of fever, nausea, vomiting, stomach cramps and diarrhea.

Zinc chloride is caustic and causes severe and occasionally fatal irritation of the epithelium lining the trachea and bronchi. Acute interstitial fibrosis of the lung occurred in one fatal case following inhalation of $ZnCl_2$ smoke from a smoke generator (Milliken et al. 1963).

Chronic administration of 0.5-34.4 mg zinc oxide per day for periods of 1 month to 1 year failed to produce signs of toxicity in rats

(Drinker et al. 1927 as cited by Stokinger 1981). In another study, 0.1% zinc was tolerated in the diet of rats, but more than 0.5% zinc reduced their capacity to reproduce, and 1% inhibited growth and caused severe anemia and death (Sutton and Nelson 1937 as cited by Stokinger 1981).

Zinc oxide has been implicated as the cause of metal fume fever in industrial situations. Typically, brass foundry workers exposed to fumes containing zinc experience an unusual metallic taste which is accompanied by dryness and irritation of the throat, with coughing and dyspnea, weakness, pains in the muscles and joints, and a high fever which is followed by profuse sweating. Recovery occurs within 24-48 hours after an attack provided exposure is discontinued. Gastrointestinal disturbances and localized dermatitis may also occur after exposure to zinc oxide.

QUANTITATIVE DESCRIPTION OF HEALTH EFFECTS

EPA (1984) derived an inhalation reference dose for zinc of 0.7 mg/day based on the American Conference of Governmental Industrial Hygienists (ACGIH 1980) recommended time-weighted average Threshold Limit Value (TLV) for zinc chloride of 1 mg/m^3. This TLV was adjusted to account for differences in the likely exposure of worker and nonoccupationally-exposed individuals, and was divided by an uncertainty factor of 10 to protect individuals in the general population who might be especially sensitive to the chemical agent. Dividing this reference dose of 0.7 mg/day by the breathing rate of the average person (20 m^3/day) gives a reference concentration for zinc in air of 35 ug/m^3.

An oral reference dose of 14.9 mg/day (0.21 mg/kg/day) was derived by EPA (1984) based on the occurrence of anemia and reduced blood copper in some patients receiving therapeutic doses of approximately 150 mg/day zinc for extended periods of time. A safety factor of 10 was used to protect sensitive individuals.

REFERENCES

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1980. Documentation of the Threshold Limit Values and Biological Exposure Indices. 4th ed. Cincinnati, Ohio

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984. Health Effects Assessment for Zinc (and Compounds). Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-048

HAMMOND, P.B. and BELILES, R.P. 1980. Metals. In Doull, J., Klaassen, C.D., and Amdur, M.O., eds. Casarett and Doull's Toxicology. 2nd ed. Macmillan Publishing, New York. Pp. 409-467

MILLIKEN, J.A., WAUGH, D., and KADISH, M.E. 1963. Can. J. Med. Assoc. J. 88:36 (As cited in ACGIH 1980)

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1975. Criteria for a Recommended Standard. . . Occupational Exposure to Zinc Oxide. Publication No. 76-104

STOKINGER, H.E. 1981. The Metals. In Clayton, G.D., and Clayton, F.E., eds. Patty's Industrial Hygiene and Toxicology. 3rd revised ed., Vol. 2A. John Wiley and Sons, New York. Pp. 1493-2060

APPENDIX PHE-3

ESTIMATED SOIL INGESTION RATES FOR USE IN RISK ASSESSMENTS

APPENDIX PHE-3

ESTIMATED SOIL INGESTION RATES FOR USE IN RISK ASSESSMENTS

SUMMARY

Assessing the risks to human health posed by contaminants present in soil often requires some information on likely soil ingestion rates. Several authors have attempted to estimate these rates, but their estimates were based primarily on limited information about the amount of soil that people get on their hands and assumptions about mouthing behavior. However, some empirical data on soil ingestion rates have recently become available. These data can be used to derive more accurate estimates of soil ingestion rates for use in risk assessments. Estimates of the most probable soil ingestion rates range from 20 to 100 mg/day, depending on the age of the individual at risk. Realistic worst-case estimates unlikely to underestimate exposure range from 100 to 500 mg/day. A value of 5,000 mg/day is considered a reasonable worst-case estimate for children with habitual pica.

Exposure is a function of both the quantity of contaminants with which one comes into contact and the duration of that contact. Therefore, the frequency of exposure is also important in determining annual exposure rates and, consequently, the risks posed by contaminated soil. This factor can only be assessed on a site-specific basis. For sites where residential areas are contaminated, exposure should be considered to be year-round. Of course, people in warmer climates will spend more time outdoors and will be exposed to larger amounts of soil than people in areas that are covered with snow for much of the year. These factors should be considered at least qualitatively in assessing exposure. In situations where contact with contaminated soil is likely to be infrequent, the exposure frequency should be estimated.

INTRODUCTION

Scientists attempting to evaluate the risks posed by contamination at a hazardous waste site must generally examine several routes of exposure. Those usually considered of most concern are:

- Ingestion of contaminated drinking water,
- Ingestion of contaminated fish and shellfish, and
- Inhalation of airborne contaminants.

These are probably the most significant routes of exposure to chemicals that are mobile in the environment. However, exposure via other routes may pose significant risks, particularly for contaminants that are quite toxic but not very mobile (e.g., polychlorinated biphenyls (PCBs), tetrachlorodibenzo- p-dioxin (TCDD), and some heavy metals). Exposure parameters such as liters of water consumed per day or cubic meters of air breathed per day have been fairly well defined for the more common routes of exposure. However, only limited work has been done to estimate these parameters for other pathways. This paper will address a potentially significant route of exposure to relatively immobile hazardous materials in contaminated soil, namely, ingestion.

PREVIOUS ESTIMATES OF SOIL INGESTION RATES

Exposure to contaminants via ingestion of soil[1] can occur by inadvertent consumption of soil on the hands or food items, mouthing behavior, consumption of nonfood items (pica[2]), or a combination of these routes. Several authors have attempted to quantify the amount of soil ingested by children. A review of these studies provides the information needed to derive a reasonable upper limit and a most probable estimate of soil ingestion rates for children and adults.

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[1] Although several authors distinguish between soil and dust in their assessments, the data do not appear to be complete enough to allow this type of differentiation. In addition, soil commonly makes up a major portion of indoor dust (Hawley, 1985). Consequently, the term "soil" will be used in this discussion to refer to soil and dust combined.

[2] See bottom of next page.

Lepow et al. (1974, 1975) measured the amount of dirt that could be removed from children's hands using an adhesive label and observed the frequency with which children mouthed nonfood objects and their hands. The average age of the children was 4.3 years. The authors measured an average of 11 mg of soil on the children's hands. They observed that a reasonable estimate of mouthing frequency is 10 times per day but that this value may underestimate the number of times children put their hands or nonfood objects into their mouths. They concluded that 100 mg/day was a reasonable estimate of the amount of soil ingested by children. They also performed several calculations using 250 mg as the amount of soil children ingest, apparently as an upper limit estimate.

Day et al. (1975) estimated that children playing outdoors could collect between 5 and 50 mg of soil on a sticky sweet. They estimated that children eat 2 to 20 sweets per day, which would lead to the ingestion of between 10 and 1,000 mg/day of soil.

Duggan and Williams (1977) attempted to determine the amount of street dust that urban children ingest daily. The authors estimated the amount of dust that was retained on the inside of the forefinger and thumb by rubbing a specified amount of dust between the fingers and reweighing the dust that was not retained. They ran several tests on different people and determined that about 4 mg (2 mg per finger) were retained. The authors then assumed that a child would suck his or her forefinger or thumb 10 times per day and concluded that 20 mg was a reasonable estimate of the amount of dust ingested daily. Duggan and Williams, (1977) also cited the estimates of Day et al. (1975) and Lepow et al. (1974) and a value

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[2] The term "pica" refers to both normal mouthing with subsequent ingestion of nonfood items, which is quite common among children at certain ages, and the unnatural craving for and habitual ingestion of nonfood items. The latter is an uncommon condition that is generally associated with medical conditions such as malnutrition, certain neurobehavioral disorders, and iron deficiency anemia or, less often, with a particular cultural background. The term "habitual pica" will be used in this paper to refer to this unusual type of ingestion of nonfood items. The term "pica" will be used for the normal ingestion of nonfood items common in children at certain ages.

from Bryce-Smith (1974 as cited in Duggan and Williams 1977) of one-thirtieth of a gram (derivation unspecified). They concluded that children probably ingest between 10 and 100 mg of street dust per day and estimated that the average daily ingestion rate is 25 mg.

Mahaffey (1977) examined sources of lead to which children might be exposed. She reported that the best clinical estimates indicate that children with pica (nonhabitual) are likely to ingest between 1,000 and 3,000 mg of paint per week. Assuming that similar amounts of soil are ingested, a child with pica would ingest between 140 and 430 mg of soil per day.

Schaum (1984) also estimated the amount of soil that children are likely to ingest. He cited the Lepow et al. (1975) estimate (100 mg/day) and also reported that habitual pica could increase ingestion levels to as much as 5,000 mg/day (Chisolm, 1982 as cited in Schaum, 1984). He determined that a range of 100 to 5,000 mg/day was reasonable for use in risk assessments.

Kimbrough et al. (1984) estimated age-specific levels of soil ingestion that the authors felt were reasonable yet unlikely to underestimate exposure (Table 1). These estimates were reportedly based on a study of lead uptake from contaminated soils.

Hawley (1985) estimated outdoor soil and indoor dust ingestion rates for three age groups: young children (2.5 years old), older children (6 years old), and adults. He based his soil ingestion estimate for very young children on Lepow et al.'s (1975) calculations of the maximum soil intake (250 mg/day), and his other soil and dust ingestion estimates on assumptions about the amount of soil or dust likely to collect on the hands and subsequently be ingested. Hawley's (1985) values for daily soil and dust ingestion are presented in Table 2; his average daily ingestion rates for soil and dust combined are also reported.

TABLE 1

SOIL INGESTION BY AGE AS ESTIMATED BY
KIMBROUGH ET AL. 1984

(mg/day)

Age	Level
0-9 months	0
9-18 months	1,000
1.5-3.5 years	10,000
3.5-5 years	1,000
Over 5 years	100

TABLE 2

SOIL AND DUST CONTACT RATES AS ESTIMATED BY HAWLEY 1985

Age Exposure Route	Medium	Ingestion Rate (mg/day)	Frequency of Exposure	Comments
2.5 year old				
Outdoors (May-Oct.)	Soil	250	5 days/wk for 5 years	Based on Lepow et al. (1975) study
Indoors (May-Oct.)	Dust	50	Daily for 6 months	Assumes ingestion of dust on inside of hands
Indoors (Nov.-Apr.)	Dust	100	Daily for 6 months	
Total	Soil and dust	164	Daily	
6 year old				
Outdoors (May-Sept.)	Soil	50	Daily for 5 months	Assumes ingestion of half the dirt on fingers
Indoors	Dust	3	Daily	Assumes ingestion of dust on inside of hands
Total	Soil and dust	24	Daily	
Adult				
Outdoors (May-Sept.)	Soil	480	2 days/week for 5 months	Assumes ingestion of dirt while smoking
Attics	Dust	110	12 times/ year	Assumes ingestion of dirt on surface of hands while in dusty attic
Living space	Dust	0.56	Daily	
Total	Soil and dust	61	Daily	

The first empirical data on soil ingestion rates were recently collected by Binder et al. (1985), who measured soil ingestion by children 1 to 3 years old using a method previously employed to measure soil ingestion by ruminants. The trace elements silicon, aluminum, and titanium, all of which are present at high concentrations in soil and none of which are absorbed by humans, were measured in the soil and in stool samples from 59 children 1 to 3 years old. The concentrations of the elements in the soil and in daily fecal samples were then compared to estimate daily soil ingestion. The authors used several assumptions in their calculations. Two of these, that dietary intake of the elements was negligible and that fecal weight was 15 g (and not the 7 g they measured) would lead to an overestimation of soil ingestion rates, possibly by a significant amount. Their assumption that the elements were entirely unabsorbed might lead to a slight underestimation of exposure. They attempted to determine the likely qualitative effects of each of these assumptions but could not estimate the magnitude of any potential effect.

The arithmetic means of the soil ingestion levels that were estimated using two of the elements, silicon and aluminum, were in general agreement (184 vs. 181 mg/day), while the mean value reported for titanium (1,834 mg/day) was an order of magnitude higher (Table 3). It appears likely that some other source of titanium in the diet was responsible for the high mean value. Therefore, the values reported for silicon and aluminum--approximately 180 mg/day—are probably more accurate. As noted above, the effect of the various assumptions on the reported values has not been quantified. However, it seems likely that the values overestimate soil consumption by the average child.

ESTIMATION OF SOIL INGESTION RATES

Soil ingestion can occur at any age but is most prevalent in young children. Baltrop (1966 as cited in Mahaffey, 1977) reported that over 75% of children 1 to 3 years old mouthed objects and that 35% ingested them. The author also noted that the prevalence of these activities decreased with age; only 33% of children 4 to 5 years old mouthed objects, and only 6% had pica.

TABLE 3

SOIL INGESTION BY CHILDREN 1 TO 3 YEARS OLD AS ESTIMATED BY BINDER ET AL. 1985

(mg/day)

Estimation Method[a]	Arithmetic Mean	Geometric Mean	Standard Deviation	Upper 95th Percentile
Aluminum	181	128	203	584
Silicon	184	130	175	578
Titanium	1,834	401	3,091	9,590

[a]In other words, elements upon which estimates are based.

SOURCE: Binder et al., 1985

Mahaffey (1977) further reported that other estimates indicate that approximately one-half of all children 1 to 3 years old have pica. Mahaffey and Annest (1985) reported that pica is most prevalent in children 6 months to 3 years old, and it is significantly more common in children from lower income families.

The estimates of soil ingestion levels reported in previous studies are summarized in Table 4. Reasonable estimates of soil ingestion by children can be derived from this information. To ensure that risk assessments based on these estimates do not underestimate risk, we will also calculate upper confidence levels.

The Binder et al. (1985) study is the only paper that presents clearly empirical data. Therefore, it is appropriate to use this study as the basis for estimating soil ingestion levels. The authors reported an average intake of 180 mg/day of soil (after excluding the results based on titanium levels) for children 1 to 3 years old. The standard deviation was approximately 200 mg/day, and the 95th percentile upper confidence limit was 580 mg/day. Binder and her colleagues assumed that none of the elements came from the normal diet, which, as mentioned above, is likely to lead to an overestimation of the amount of soil ingested. Consequently, a value of 500 mg/day, which is around the 95th percentile upper confidence limit of the arithmetic mean values reported, would appear to be a conservative yet reasonable estimate of soil ingestion for children 1 to 3 years old. As noted above, older children are less likely to exhibit pica. However, Lepow et al. (1974, 1975) observed children 2 to 6 years old mouthing objects, and it is probably reasonable to use this value of 500 mg/day as a worst-case estimate of soil ingestion for children 1 to 6 years old. A value of 100 mg/day, which is near the geometric mean of Binder et al.'s (1985) data, appears to be a reasonable estimate of the most probable ingestion rates.

Children less than 1 year old are not likely to come into direct contact with soil regularly. However, very young children may be exposed to contaminated house dust. No specific information is available on ingestion

TABLE 4

ESTIMATED LEVELS OF SOIL INGESTION
REPORTED IN THE LITERATURE

(mg/day)

Reference	Age	Level	Comments
Lepow et al., 1974, 1975	4.3 years (mean)	100	Based on observations of mouthing behavior; (see text)
Day et al., 1975	Children 1-3 years	10-1,000	Based on ingestion of soil on candy
Duggan and Williams, 1977	Children 2-6 years	25	See text
Mahaffey, 1977	1-3 years	140-430	Based on estimate of paint consumption by children with pica
Schaum, 1984	2-6 years	100-5,000	See text
Kimbrough et al., 1984	0-9 months 9-18 months 1.5-3.5 years 3.5-5 years Over 5 years	0 1,000 10,000 1,000 100	Based on study of lead exposure; probably overestimates risk
Hawley, 1985	2.5 years 6 years Adults	164 24 61	Based on estimated ingestion of both and dust; see text
Binder et al., 1985	1-3 years	180±200	Based on measurement of trace elements; see text

of dust by children in this age group, and we suggest a value of half the previous value, or 250 mg/day, as a reasonable, worst-case estimate of ingestion levels. Similarly, 50 mg/day is probably a reasonable estimate of the most probable ingestion rate for children in this age group.

No empirical information on older children was available. Mahaffey (1977) reported that mouthing decreased in most children more than 4 years old. It seems reasonable to assume that almost all children 6 to 11 years old would have reduced their soil ingestion by at least 50%. Consequently, we suggest that 250 mg/day is a worst-case estimate and 50 mg/day, a most probable case estimate of soil ingestion by children in this age group.

For children more than 11 years old and adults, the value of 100 mg/day recommended by Kimbrough et al. (1984) appears to be a reasonable, worst-case estimate of daily soil ingestion. Based on Hawley's (1985) assumptions, 50 mg/day appears to be a reasonable estimate of the most probable ingestion rate for people who have frequent hand-to-mouth contact (e.g., smokers) or who are in direct contact with contaminated soil (e.g., construction workers, gardeners). A value of 20 mg/day appears to be a reasonable estimate of the most probable soil ingestion rates for adults who are not smokers and do not regularly engage in outdoor activities.

As noted earlier, certain individuals exhibit pica habitually, i.e., they eat nonfood items regularly, even daily. The values for soil ingestion reported above may underestimate exposure for people exhibiting this type of behavior. The upper limit on soil ingestion of 5,000 mg/day recommended by Schaum (1984) probably is a reasonable, worst-case estimate of soil ingestion by a person with habitual pica.

Our estimates of soil ingestion by persons in different age groups are presented in Table 5. These values fall within the range of soil ingestion levels suggested by Schaum (1984) but are generally lower than the values presented by Kimbrough et al. (1984). However, it should be noted that these estimates are in general agreement with the estimated levels of soil ingestion presented in Table 4. Consequently, we feel that these

TABLE 5

ESTIMATES OF SOIL INGESTION RATES

Age	Average Weight[a] (kg)	Worst Case[b] (mg/day)	Most Probable Case (mg/day)
0-1 years	10	250	50
1-6 years	15	500	100
6-11 years	30	250	50
Over 11 years	70	100	50[c]

[a]EPA, 1985

[b]This does not include individuals who exhibit habitual pica. For them, the upper value presented in Schaum (1984) of 5,000 mg/day would be more appropriate.

[c]A value of 20 mg/day is probably a more reasonable estimate of soil ingestion rates for adults who do not exhibit frequent hand-to-mouth activity (e.g., most nonsmokers) and do not regularly engage in outdoor activities.

values are reasonable estimates that are unlikely to underestimate soil ingestion levels. Therefore, they are unlikely to lead to any underestimation of exposure via this route.

ESTIMATION OF EXPOSURE FREQUENCY

Several authors estimated the frequency of exposure to soil. Kimbrough et al. (1984) assumed that people would only come into contact and ingest soil for 6 months each year. Schaum (1984) noted that the ground is frozen for an average of 118 days/year in the coldest parts of the United States, while in other parts, people can come into contact with outdoor soil year-round. He therefore determined that exposure could occur for anywhere between 247 and 365 days/year. Hawley (1985) noted that people are likely to be exposed year-round to soil, both outdoors as soil and indoors as a major component of dust. Consequently, a reasonable, worst-case assumption appears to be that people come into contact with and ingest soil year-round.

Clearly, site-specific factors should be considered if in estimating likely contact and ingestion rates. It usually is not possible to determine the effect of these factors on the rates of exposure to soil quantitatively. However, any site-specific factors (such as climate) likely to affect the frequency of exposure should be addressed qualitatively.

This discussion of exposure frequency only applies to inhabitants of contaminated residential areas or to people living immediately adjacent to a source of contamination. In the case of people (children or adults) who infrequently use a contaminated area, a site-specific estimate of the frequency of exposure must be derived.

CONCLUSION

No single value will accurately reflect daily soil ingestion rates for all individuals. Too many factors can influence contact with soil and consequently affect the quantity of soil ingested. However, risk managers often need to estimate average soil ingestion rates in order to assess

potential exposure to soil-bound contaminants. The values for soil ingestion rates presented in this paper have been derived using the best information currently available. As such, they provide reasonable, yet conservative (unlikely to underestimate exposure), estimates for use in the risk assessment process.

REFERENCES

BINDER, S., SOKAL, D., and MAUGHAN, D. 1985. Estimating the amount of soil ingested by young children through tracer elements. Draft. Centers for Disease Control, Atlanta, Georgia, July 31, 1985.

DAY, J.P., HART, M., and ROBINSON, M.S. 1975. Lead in urban street dust. Nature 253:343-345.

DUGGAN, M.J., and WILLIAMS, S. 1977. Lead-in-dust in city streets. Sci. Total Environ. 7:91-97.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985. Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessments. Office of Health and Environmental Assessment, Washington, D.C. March 1985. OHEA-E-161.

FORD, K.L., and GURBA, P. 1984. Health risk assessments for contaminated soils. In The Proceeding of the 5th National Conference on Management of Uncontrolled Hazardous Waste Sites. Washington, D.C., November 7-9, 1984.

GALLACHER, J.E.J., ELWOOD, P.C., PHILLIPS, K.M., DAVIES, B.E., and JONES, D.T. 1984. Relation between pica and blood lead in areas of differing lead exposure. Arch. Dis. Child. 59:40-44.

HAWLEY, J.K. 1985. Assessment of health risk from exposure to contaminated soil. Risk Analysis 5:289-302.

KIMBROUGH, R.D., FALK, H., STEHR, P., and FRIES, G. 1984. Health implications of 2,3,7,8-tetrachlorodibenzodioxin (TCDD) contamination of residential soil. J. Toxicol. Environ. Health 14:47-93.

LEPOW, M.L., BRUCKMAN, L., RUBINO, R.A., MARKOWITZ, S., GILLETTE, M., and KAPISH, J. 1974. Role of airborne lead in increased body burden of lead in Hartford children. Environ. Health Perspect. 7:99-102.

LEPOW, M.L., BRUCKMAN, L., GILLETTE, M., MARKOWITZ, S., RUBINO, R., and KAPISH, J. 1975. Investigations into sources of lead in the environment of urban children. Environ. Res. 10:415-426.

MAHAFFEY, K.R. 1977. Quantities of lead producing health effects in humans: Sources and bioavailability. Environ. Health Perspect. 19:285-295.

ROELS, H.A., BUCHET, J.P., LAUWERYS, R.R., BRUAUX, P., CLAEYS- THOREAU, F., LAFONTAINE, A., and VERDUYN, G. 1980. Exposure to lead by the oral and the pulmonary routes of children living in the vicinity of a primary lead smelter. Environ. Res. 22:81-94.

SCHAUM, J.L. 1984. Risk Analysis of TCDD Contaminated Soil. Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Washington, D.C. November 1984. EPA 600/8-84-031.

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